ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 51

Published Quarterly

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE . 1010 BEAUMONT BLDG.
BUSINESS OFFICE . 7200 WYDOWN BL.

ST. LOUIS, MO., U.S.A.

Annual Subscription in United States, Spain, Central and South America, $6.00 in Advance. Canada, $6.40. Other Countries, $6.80.

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OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 51 JUNE, 1942 No. 2

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OTOLARYNGOLOGICAL PROBLEMS OF AVIATION IN WORLD WAR II*

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For the second time within the life span of all and within the period of otolaryngological practice of many our nation has been called upon to defend its way of life and its conception of things worth living for. This same era also has supported the growth of air power from an obstreperous infant to a gargantuan monster capable of destroying or—depending upon the magnitude of their effort—of defending those to whom it owes its creation. Air power today comprises four elements; the man, the machine, supply and maintenance coordinated in such a manner that they may operate as a single unit. The man is the pivot about which the other elements operate and is of course the most important of the group. As man is called upon to function under conditions somewhat remote to the intent of the forces which guided his evolutionary processes many physiological problems have arisen, a large share of which embrace otolaryngology.

The wisdom and vision of those charged with our destiny at the time of our entrance into World War I fostered a broad understanding of the otolaryngological aspects of flight. As a conse-

*Read before the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., June 2, 1942.

quence, otolaryngologists had their share in the guidance of much of the earlier development of aviation medicine. Several of these pioneers today are members of the "Triological" Society. It would be a presumption upon my part to attempt to describe their work which is so well known to all of us.

Aviation of World War II has been characterized by widespread increase in speed, altitude of operation, motor power, and numbers of men involved. These changes had their influence upon our problems. I have attempted to list these problems in order of their importance. They are: (1) selection; (2) the effect of flight upon hearing; (3) aero-otitis media; (4) airsickness; (5) aero-sinusitis; (6) the effect of altitude on the voice; (7) the effect of anoxia upon the hearing mechanism.

It is obvious that many of these problems are similar to those studied twenty-four years ago, but in some cases their relative position in the scale of importance has been altered. Today aircraft speed is more than three times that of World War I. Ceilings are more than twice as high and the motor power used at times may be more than ten times as great.

The tremendous numbers of men necessary to form air crews for the present Air Force expansion program has relegated the problem of selection to the position of greatest importance. In January of this year the President announced that 50,000 combat planes would be produced in the United States in 1942 and 100,000 in 1943.[1] Each air crew consists of from one to seven men or more depending upon the size and type of aircraft. Thus the program requires a vast number of men, possibly taxing the available qualified man power of the nation. Thus we can no longer seek supermen. It can be readily demonstrated that a large proportion of average intelligent healthy normal individuals through intensive training, can be taught to master all of the intricacies of flight and combat duty in a reasonable length of time. From the point of view of our specialty, a candidate can become a good airman if he simply possesses an average nose of normal function, average ears and hearing, and eustachian tubes and sinuses without obstruction. His balance mechanism must be within the normal range and he must be without danger of vertiginous episodes. Experience has proven these requirements to be adequate. Through training he can be taught to ventilate his middle ears in rapid descent. He can learn what illusions to expect from his balance mechanism and to rely upon instruments for spatial orientation. He can be admonished not to fly with nasopharyngitis or sinusitis. Although often

a skeptic he will become a firm believer after his first violation of that axiom.

As otolaryngologists our second great problem today is to help the air crew to hear well and to keep their hearing at a high degree of efficiency at all times. The information gained through their ears is absolutely necessary for operational flight. The radio beam is now the roadway of the air. The crossing of beams is the milepost. By intelligent use of this adjunct and instruments for operational orientation an air crew can fly great distances without ever seeing the surface of the earth. They can check distances, ground speeds, fuel consumption, recognize their destination, drop down through the overcast and land upon an airdrome clothed in darkness or mist with a great degree of accuracy. They are also dependent upon hearing for the ever-important weather reports and for interplane communication. It is only through hearing that they are able to detect changes in motor rhythm and, as in the case of multimotored aircraft, keep the motors synchronized.

In spite of dependence upon the auditory sense, changes in altitude and fatigue of the organ of Corti produced by the 80 to 110 decibels or more of noise to which they are subjected result in a constant alteration in the curve of hearing.[2] Actually this variation takes place minute by minute. Changes in altitude resulting in a barometric pressure change of 100 mm. of mercury—about 4,000 feet at near ground levels—can between the acts of swallowing produce a fluctuation of several decibels of hearing in the conversational frequency range. Aircraft noise can produce a 15 decibel loss in the 3,000 to 5,000 frequency area in one hour's time and in my experience a 25 decibel loss after four hours of flight. Fortunately most hearing changes are transitory and after complete middle ear ventilation and a few hours of quiet depending upon the intensity of the noise and length of the exposure, usually return to normal. However, after sufficient insult, permanent changes may occur. Many of us are now called upon to analyze audiograms of airmen. It is my belief that the audiogram of this group must be interpreted in accordance with the following conception:[3]

First of all he is of certain age which has made its impression upon the higher frequencies of the audiogram. Second, during his lifetime he has been exposed to a certain amount of noise, much of which may have been from aircraft. This factor has its greatest influence upon the frequency area from 3,000 to 5,000. Third, changes in barometric pressure may have had their influence through the entire range of frequencies. The ears which he inherited, the

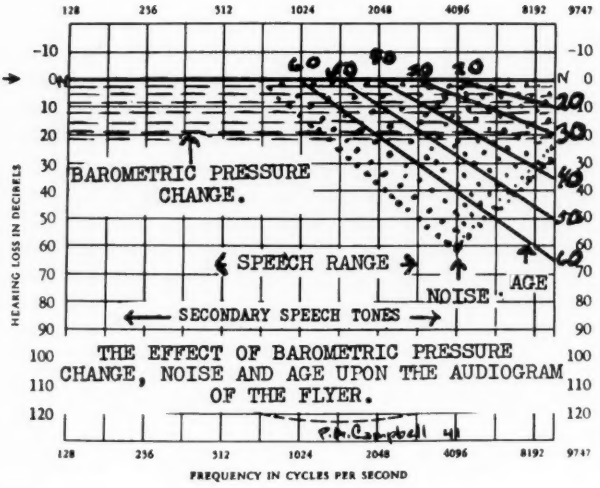

Fig. 1.

various ear diseases which he had during his earlier life must also be considered.

Aero-otitis media, as described by Armstrong and Heim,[3] is the third great problem. The mechanism by which aero-otitis media is produced is best shown in Fig. 2. Little difficulty is experienced in ascent as the air present in the middle ear and mastoid structure seeps through the eustachian tube as soon as the pressure increases to a level of 10 to 20 mm. of mercury above that of the outside air. This seepage takes place either during or between the acts of swallowing. At near ground levels 10 to 20 mm. of mercury pressure differential represents roughly from 350 to 750 feet of altitude. Upon descent, however, the conditions are quite different as most individuals are able to equalize the then relative negative pressure of the middle ear pneumatic system only through the act of swallowing or some related maneuver such as yawning or the thrusting of the jaw forward as in a stifled yawn. The mechanism of this flutter valve effect is not entirely understood. It is believed to be due to the relative differential pressure forcing together tightly the epithelium of the lumen of the eustachian tube. If through failure to swallow or through some anatomical condition or swelling of

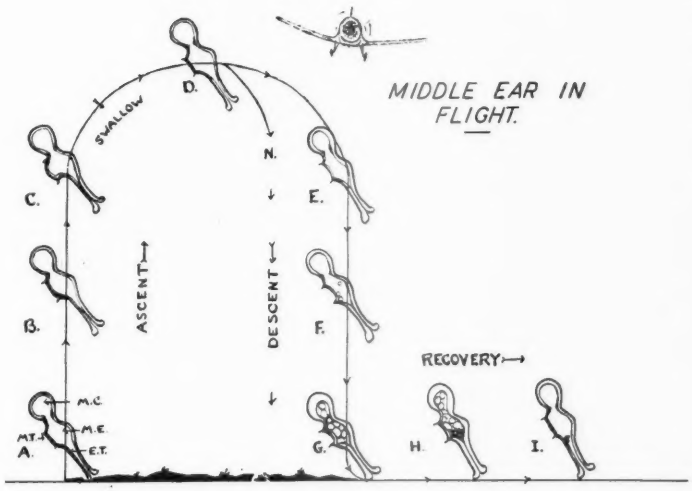

Fig. 2.—Diagrammatic representation of the effect of flight upon the middle ear. *M.T.* Tympanic membrane. *M.E.* Middle ear. *E.T.* Eustachian tube. *M.C.* Mastoid cavity. *A, B* and *C.* Ascent, during which pressure becomes relatively greater inside the middle ear than outside. The drum bulges outward during the ascent. *D.* Equalization takes place through the act of swallowing. The drum returns to normal position. *N.* Descent to earth with normal equalization. *E, F* and *G.* Descent with closed eustachian tube. The middle ear pressure is then relatively negative, and the drum pressed inward. Eustachian tube flutter valve is formed. *G. H.* and *I.* Recovery through space filling fluid production into the middle ear cavity with release of flutter valve when the pressure is equalized.

the epithelial lining such as is encountered in pharyngitis, obstruction takes place and a relative negative pressure of from 100 to 200 mm. of mercury is reached, the epithelial lining is locked tightly together. As the pharyngeal muscles act only upon the orificial end of the tube, the act of swallowing prevents the maneuver from bringing about ventilation. A cycle of events is then initiated resulting in aero-otitis. When the negative pressure inside the middle ear reaches 100 to 120 mm. of mercury the pain rather suddenly becomes quite severe, 10 or more decibels of hearing is lost usually in some portion of the low tone range (128 dvb. to 2048 dvb.) varying among individuals. On examination the drum is seen to be sharply retracted and there is engorgement of the vessels

along the handle of the malleus. A few minutes later ecchymosis may appear in Shrapnell's membrane and along the lower aspect of the handle of the malleus. After a period of from one-half hour to eight or ten hours depending upon the degree of differential pressure, serosanguineous fluid and bubbles form in the middle ear.

The formation of space-filling fluid from the mucosal lining initiates the beginning of the recovery cycle as the relative negative pressure or vacuum is reduced in proportion to the fluid which forms. After a period pressure is equalized and the flutter valve released. The structures of the ear which may have been altered to some extent by this barotrauma may take from three to twenty-one days for complete recovery. Fortunately only a very few become infected. Unfortunately, in my experience at least, Politzerization and catheterization are of little value unless performed immediately and then are not always efficient. The most efficient treatment is for the individual to return to the level at which the block occurred either in actual flight or in a pressure chamber, and then to return to ground level very slowly swallowing frequently on the descent. In this manner both the pressure outside of the drum and the pressure of the air passing through the eustachian tube are altered simultaneously whereas in catheterization or Politzerization only that entering the eustachian tube is altered. If aero-otitis has existed over a period of a few hours the only other alternative is to use heat to increase fluid production in the middle ear, analgesics for relief of pain, and shrinking medication to the orifice of the eustachian tube. Paracentesis, I believe, is absolutely contraindicated.

At this point it is timely to describe a complication which may arise from flight during even mild external otitis infections. In certain of the warm climates or during the warmer weather in most climates external otitis from various forms of mycosis or low-grade organisms such as staphylococcus is quite common. During flight the excursions of the ear drum—that is the bulging and retraction which occur during changes of altitude and during the acts of ventilation—are much greater than those encountered in everyday life. This movement seems to enhance the spread of infection first through the outer layer of the drum and then between the middle and outer layers of the drum much like a sheet of water between two panes of glass. The appearance is first that of bullous myringitis then a diffuse layer of pus between the layers forms. Fortunately we have seen no perforations. I have examined the literature and have found no reference to this phenomenon.

To turn to airsickness—operational flight during World War II is taking place in all types of weather. Even in smooth air there is more or less constant change of direction of motion. Warm air currents from dry fields, beaches, etc, flow upward forming so-called vertical gusts. The cooling effects of water, forests, vegetation, etc., cause currents to flow downward in the air pocket. Winds are deflected upward from inclines and downward from declines. Changes in weather have their effect. All of these factors bring about unpredictable turbulence of the atmosphere. Similar to that which takes place on shipboard, those individuals in the areas of an airplane most remote from the center of rotation are most effected. Thus certain members of the air crew, for instance the tail machine gunner is subjected to all of the elements which lead to airsickness. At this time there is no single simple remedy which can be used to alleviate completely this condition and yet leave the individual at maximum efficiency. True, there is a certain amount of conditioning to airsickness, but our best protection remains in the realm of selection.

Aerosinusitis has increased in frequency as greater altitudes and speeds of ascent have been reached. The mechanism of its production is similar to that of aero-otitis. In a sinus obstructed by polypi or redundant tissue, the atmospheric pressure changes cannot be equalized. On ascent the pressure inside an obstructed sinus becomes greater than that outside with consequent expansion to the limit of elasticity of its walls or structures. Pain results. During descent the opposite effect is present. Redundant tissue or polypi may then act to form a ball valve over an ostium or pus flowing over an ostium can be drawn into a sinus which may have previously been empty and sterile. If the relative negative pressure inside a sinus becomes great enough the mucosa may be torn away from the walls. Hemorrhage into the cavity or hematoma beneath the mucosa is not at all rare. Those with chronic sinusitis should not be selected for air crew duty. Those with acute sinusitis or rhinitis should not fly.

As greater altitudes are achieved more and more effect upon the quality of the voice is becoming apparent. Operational flights are becoming more or less routine at altitudes of 35,000 feet. At this level, the barometric pressure is 178 mm. of mercury or in other words, the air passing between the vocal cords is less than one-fourth as dense as at sea level. Thus the effort for the production of intelligible speech is increased as the individual attempts to compensate for the lowered density of the air by increasing the force of the diaphragmatic excursion and muscular pressure used in pressing air

upward through the larynx. Any physical effort at great altitude is extremely fatiguing. At these levels simple conversation becomes a task. The quality of the voice is also altered as the density of the air in the resonating column and cavities is decreased. These phenomena will be enhanced by further increase in the altitudes to which man may go. Only a prophet can predict what that level may be.

Our seventh problem is the effect of anoxia upon the hearing mechanism. I had hoped by the time of this meeting to have some very definite information for you as we have made several audiograms in a pressure chamber at high altitude under conditions of anoxia. However, to date, the audiograms have not demonstrated enough change or pattern to make a definite picture. As you know anoxia affects the higher centers and in some respects is similar to acute alcoholism. Consequently the problem of interpretation of sounds is difficult. We are therefore making arrangements to change over to audiometry of spoken words rather than pure tones in order that the effect of interpretation may be brought out. Possibly some time in the near future I can give you more information along this line. There are also other mechanical effects concurrent with audiometry at high altitude as rarefication of air between the condenser plates and in other portions of the electrical system will undoubtedly alter results. I feel, however, the whole problem is of great interest and will become more important as higher altitudes are reached.

In conclusion I may say that all people who are doing research directed toward the problem of making man fly higher, faster, and with more comfort and safety are very receptive to ideas which may aid them in their work. This is *our* war. Some simple mechanical device may alter its entire course. To date the genius of the American people has not been found wanting.

THE SCHOOL OF AVIATION MEDICINE.

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XXIX

CLINICAL AND EXPERIMENTAL STUDIES WITH SULFAPYRIDINE AS A HEMOSTATIC AGENT*†

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In the early months of 1940 orthopedic surgeons were using sulfamethylthiazole powder locally in the treatment of osteomyelitis. Otolaryngologists, too, were using the drug against osteomyelitis of the frontal bone, and amounts of the drug varying between 2 and 5 gm. were instilled in the operative wound after radical external frontal sinusostomy in the hope of decreasing the incidence of frontal osteomyelitis.

In the changing of dressings after such procedures, a distinct difference between the condition of treated wounds and the condition of untreated wounds was noted. It was discovered that in wounds in which, ordinarily, considerable postoperative oozing would have occurred, with subsequent saturation of the dressings, treatment with sulfamethylthiazole powder caused them to remain almost uniformly dry. This observation prompted the thought that the drug might possess hemostatic powers of some degree.

A short time thereafter an occasion for test of this hypothesis arose. A patient was admitted to the hospital bleeding from the left tonsillar fossa, six days after tonsillectomy. The bleeding was of a persistent, oozing type and had failed to respond to the usual methods of control, such as sedation, cleaning of the fossa, local application of pressure, and the application of snake venom to the fossa. The bleeding arose in the lower pole of the tonsillar fossa, close to the base of the tongue. The fossa was wiped dry and a small amount of sulfamethylthiazole powder was applied lightly with a sponge. A thin yellow scum formed on the surface of the tissue and the oozing ceased almost immediately.

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*Abridgement of thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of M.S. in Otolaryngology.

†The sulfapyridine used in these studies was supplied by Eli Lilly & Co.

‡Fellow in Otolaryngology, the Mayo Foundation.

In recent years a voluminous literature on the chemotherapeutic drugs, their properties, and their actions has accrued. Vinson[1] noted that the local application of sulfanilamide powder seemed to be effective in stopping the bleeding occasioned by dry bronchiectasis. He gave no specific information as to any possible hemostatic effect of the drug.

Methods for the control of tonsillar bleeding at present are almost as numerous as writers on the subject. Before a discussion of the methods of control is started, it might be wise to define various types of tonsillar bleeding. Myerson[2] classified tonsillar hemorrhage as primary, recurring, and secondary. Primary hemorrhage is defined as bleeding which occurs at the time of operation and continues unchecked after the patient leaves the operating room. Recurring hemorrhage is that which appears soon after the operation. Secondary hemorrhage is that which occurs between one and fourteen days after operation.

It seems obvious that primary and recurring hemorrhages may be controlled best, first, by the careful taking of a history and examination preoperatively and then, at the time of operation, by thorough tying off of all bleeding vessels.

In the case of secondary hemorrhage, a different set of factors is said to operate. First, by virtue of the fact that the patient has experienced neither primary nor recurring hemorrhage, many of the more common causes of bleeding can be excluded, such as blood dyscrasias, deficiency of vitamin C, incomplete tonsillectomy, the presence of acute inflammation at the time of operation, hypertension and arteriosclerosis, and anomalous blood vessels, as being factors in themselves causative of secondary bleeding. Secondary hemorrhage may be due to the presence of infection in the wound, or to sudden emotional or physical strain; whether or not these factors always operate seems uncertain. Occasionally, a patient who has had an uneventful postoperative course will suddenly and without apparent reason begin to bleed from the fossa on perhaps the sixth or seventh postoperative day. Frequently it is most difficult to stop this type of hemorrhage, and it may continue for as long as seventy-two hours. Such bleeding usually does not arise from a specific vessel, but is capillary in nature and occurs over a wide area, often in the region of the lower pole of the tonsillar fossa.

That secondary tonsillar hemorrhage can result in death is attested to by many reports in the literature.[3-7] The usual methods of control of secondary bleeding are (1) adequate sedation (usually

¼ gr. [0.016 gm.] of morphine sulfate by hypodermic injection), (2) cleansing of all clots from the fossa, and (3) the application of pressure in the fossa. If these measures prove to be unsuccessful, many suggested procedures are available; they range from the use of lemon juice in the fossa to sewing of the pillars together. Among the most effective of these methods is the use of derivatives of brain and lung tissue, and blood platelets. Yet no method has been totally successful. Thus, it would seem that any simple, safe, and effective method for the control of secondary bleeding should be definitely worthwhile.

An attempt was made to confirm the early observations made in respect to the effects of sulfonamide products used for this purpose. A group of seven patients (including the patient already mentioned) was used, and the drugs were applied locally to the bleeding tonsillar fossae after the ordinary methods, such as sedation, cleaning of the fossa, and pressure, had failed to produce lasting hemostasis. First, however, results of experimental studies will be described.

STUDIES IN THE EXPERIMENTAL LABORATORY

Since cases in which clinical observations could be made were necessarily few in number, it appeared that an experimental study in the laboratory would present an opportunity for observation of the action of the drugs as applied to oozing surfaces in a large number of cases.

Throughout the experiments, powdered sulfapyridine, sulfanilamide, sulfathiazole, and sulfamethylthiazole were used. Untreated or talc-treated wounds served as controls. In the first series of experiments a total of fifty-six guinea pigs was used. Circular wounds 1.5 cm. in diameter were placed one on each side of the back of the animal at points equidistant from the midline. Wounds were made while the animals were under the influence of anesthesia with ether, and the animals were then returned to their cages without the taking of special precautions against infection. Five to seven days later the scabs which had formed over the wounds were evulsed, so that an open surface remained, granulating and oozing freely. If a wound was found to be badly infected, it was not employed in the experiment.

After evulsion of the scabs, the oozing wounds were sprayed with the various powdered drugs, a powder blower being used. Wounds were sprayed with powder until they were thoroughly and

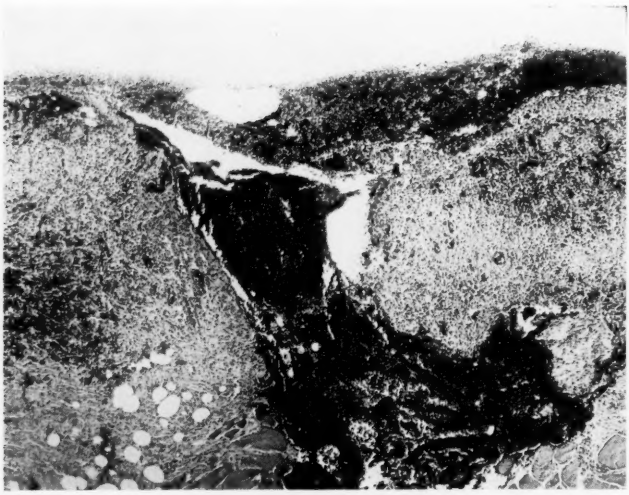

Fig. 1.—Section taken from untreated wound five minutes after removal of scab, as described in text. Note abundance of free erythrocytes near and on the surface (x45).

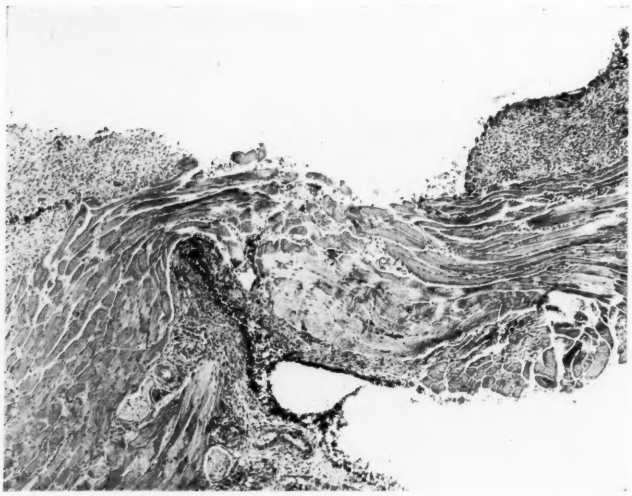

Fig. 2.—Section taken from a wound similar to that in Fig. 1, treated with sulfapyridine powder. The surface of the wound shows no evidence of bleeding. Some evidence of bleeding can be seen on the untreated surface of tissue taken in section (x45).

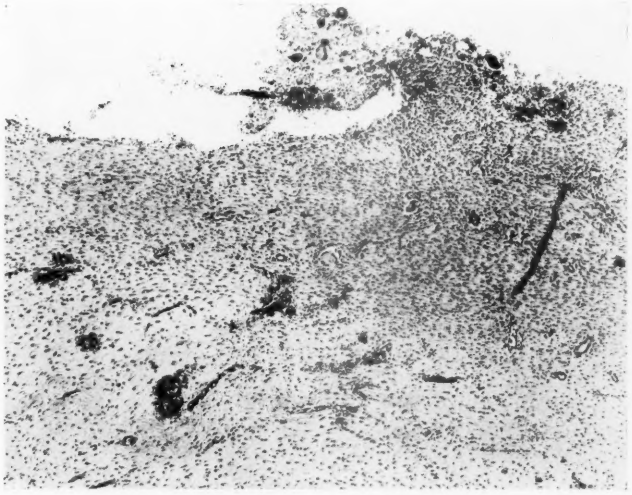

Fig. 3.—Section taken from similar wound treated with sulfamethylthiazole powder. No evidence of bleeding is seen on the surface of the wound (x45).

smoothly covered. An effect, if one occurred at all, was noted immediately, but it was found that if the surface of the wound was gently rubbed within three to five minutes oozing might recur; after five minutes had elapsed this manipulation had no effect, if once the bleeding had been thoroughly stopped.

In this series of experiments it was found that wounds treated with talc continued to ooze unabated; sulfanilamide and sulfathiazole likewise had no noticeable effect on this oozing. When sulfamethylthiazole was sprayed on the surface of the wound, however, an immediate effect was noted; a yellowish scum, closely adherent to the surface of the wound, seemed to form. Oozing simultaneously ceased and the wound remained dry unless this surface layer was rubbed from the wound; if this was done, bleeding started again.

Wounds sprayed with powdered sulfapyridine responded at least as well as, and often better than, those sprayed with sulfamethylthiazole powder. The technic consisted of the spraying of a fine layer of powder over the surface of the wound. Bleeding and oozing usually were affected immediately, the surface of the

wound becoming rather dry and covered with a thin, tenacious coating of the drug. This coating could not easily be dislodged.

Sections taken from the treated wounds three to five minutes after application of the drugs to the wounds differed in only one respect from sections taken at the same time from untreated or talc-treated control wounds. In sections made of untreated wounds there were usually many free erythrocytes on the surface and about the ends of blood vessels; in the treated wounds free erythrocytes were notably absent (Figs. 1, 2 and 3).

Cultures were made of material taken from several of the wounds twenty-four hours after treatment; the results were uniform in that in many cases talc-treated or untreated control wounds remained infected; the decrease in infection in those wounds treated with any of the four chemotherapeutic agents was dramatic (Fig. 4).

In four groups of experiments the horse was utilized as the experimental animal. According to the technic previously described, the sides of the neck of the horse were clipped close with the fine animal clipper, and wound areas 2.5 cm. in diameter were marked out, six on each side of the animal's neck, spaced at intervals of 4 cm. between each wound site. Anesthesia of the area by infiltration with procaine hydrochloride was then produced and circular wounds were made with scalpel and scissors, according to the standard wound outlines. The skin and subcutaneous fascia were removed and the underlying muscle was cut into. If large vessels were encountered, they were ligated; otherwise the wounds were allowed to bleed freely and to start to heal without treatment. The animals were then allowed to continue their normal way of existence.

Six days later, the scabs were evulsed from the wounds by the gentle insertion of a scalpel at the edge of the wound area, followed by grasping of the free edge of the scab with a forceps and swift removal of the entire scab. If uninfected, these wounds then bled and oozed freely from a fresh bed of granulating tissue. Each of our finely ground powders (sulfanilamide, sulfapyridine, sulfathiazole, sulfamethylthiazole, and talc) was then sprayed evenly over the bleeding surface of a wound, one wound being left untreated as a control in addition to the talc-treated control. Much the same result was noted in these experiments as had been noted in the work with guinea pigs, except that the effect was more noticeable, if anything, in the wounds of the horses. The wounds sprayed with sulfanilamide, sulfathiazole and talc, as well as the wounds left un-

Fig. 4.—Lower part of illustration shows culture of organisms from a control wound that had been treated with talc; note complete hemolysis on blood agar plate. The left upper section shows culture of organisms taken from wound treated with sulfapyridine; the only growth evident here apparently is due to contaminants. The right upper section shows culture of organisms taken from wound treated with sulfamethylthiazole; the only growth here apparently is due to contaminants.

sprayed, oozed for from five to ten minutes. The wounds treated with sulfapyridine and sulfamethylthiazole exhibited an immediate tendency toward hemostasis.

The wounds sprayed with sulfamethylthiazole became covered with the usual yellow scum which could not be separated from the tissues thereafter. Simultaneously, the surface of the wound appeared to be dry. The sulfapyridine-treated wounds responded in much the same manner, except that the surface of the wound, after being sprayed with sulfapyridine, was covered with a white fine powder and beneath this the surface of the wound seemed to become rather hard and very dry. Oozing usually was controlled almost immediately. The subsequent appearance of the wounds as they healed was of interest. About those wounds treated with any of

the chemotherapeutic agents there were much less edema and inflammatory reaction than about the talc-covered and the untreated wounds. These latter usually exhibited unmistakable signs of infection during the second period of healing. No wounds were so badly infected in this series that they could not be utilized, although perhaps 30 per cent of the wounds exhibited mild infection, which disappeared rapidly after the local application of the chemotherapeutic powders.

All observations thus far necessarily had been made grossly, because the use of powders in the wounds made impossible any attempt at observation of the wound itself. It therefore seemed logical to try sodium sulfapyridine and observe its effect on bleeding surfaces, since if this was useful, a solution of it could be employed and the wounds could be viewed with the aid of the dissecting microscope. A group of four guinea pigs was used, and two wounds were made on each, in the manner previously described. After six days the scabs were removed from the wounds; all of these wounds appeared to be clean and were healing well. The granulations oozed freely. Powdered sulfapyridine was applied to one wound on each animal and sodium sulfapyridine was applied to the other. The sulfapyridine-treated wounds became dry almost at once. The wounds treated with sodium sulfapyridine powder did not become dry immediately, due possibly to the hydroscopic properties of the drug. The surfaces of the wounds, however, appeared to be covered by sludge, so to speak, and oozing was not sufficient for the blood to seep out over the edges of the wound in any instance. After approximately a five-minute interval, the wounds appeared to be fairly dry; at the end of a fifteen-minute interval both groups of wounds had about the same appearance, both being dry and clean.

A 40 per cent solution of sodium sulfapyridine was next tested. Four wounds were made on two guinea pigs in the usual manner. Only that amount of the solution which would thoroughly cover the oozing surface was placed on the wound. Within three to five minutes the wounds appeared to be uniformly dry, and it was noted that the base of each wound was covered with what appeared to be a fine, silver-like precipitate. The wounds remained dry, and at the end of a half-hour were still dry and covered by the film, which by this time had almost the consistency of scab formation.

The next experiment consisted in observation of the action of an aqueous solution of sodium sulfapyridine on an open wound, under the dissecting microscope. A white rabbit was thought to

be best for this group of experiments because of the relative ease with which the superficial blood vessels on the abdomen of this animal can be observed. An attempt was made to create again a standard technic, and it was found that suitable wounds could be produced by using a large white rabbit, placing it securely on its back with the aid of an animal board, and carefully clipping the fur from most of the abdomen. It was then possible to mark out two areas 4 by 2.5 cm., placed equidistant from the midline and apparently having a similar blood supply. These were scarified and two abraded surfaces were produced, in which free oozing from the capillaries and small vessels occurred. The one wound was kept moist by means of physiologic solution of sodium chloride; the other, with a 40 per cent solution (aqueous) of sodium sulfapyridine over a thirty-minute period. At the end of this time the wound on which the solution of sodium chloride had been used was still moist, and the surface was still raw and oozing slightly. On the other abraded surface, treated with the solution of sodium sulfapyridine, a change was noted in the appearance of the wound about two minutes after its application, and a very fine silver-appearing precipitate seemed to form over the surface of the wound. As in previous experiments, this could not be dislodged from the tissues. The wound then blanched unevenly, and there was no further sign of oozing or of open, small vessels; the unbroken skin vessels did not seem to change in appearance however, and blood still could be observed in them, as in the other wound and as before the application of the drug. At the end of the thirty-minute period the wound was allowed to dry and it immediately appeared to be crusted over by a hard, light brown scab, which was not present on the control wound. This experiment was repeated three times, similar results being obtained each time.

Since the hydrogen ion concentration of the sodium sulfapyridine solutions used was about 11, it was necessary to consider the possibility that the action of the drug might be due to its extreme degree of alkalinity. An experiment was therefore arranged which simulated that previously described, except that a one-tenth normal solution of sodium hydroxide was used instead of the physiologic solution of sodium chloride of the previous experiment. The reaction of the wound treated with sodium sulfapyridine to this last solution was the same as the preceding reactions. In the sodium hydroxide-treated wound some degree of oozing continued throughout the experiment, although thirty minutes after termination of the experiment (one hour after the beginning) the surface of the wound began to feel dry and hard.

Another experiment was then conducted in which a 40 per cent solution of sodium sulfapyridine was used as before, with a control of physiologic solution of sodium chloride, but this time in a heparinized animal. Preparation of the wounds proceeded as previously described, except that the inner aspect of the hindleg was used as the site of the wound on either side. At the start of the experiment 2,000 units of solution of heparin (0.2 c.c.) were injected into a vein of the right ear of the rabbit, and the administration of 1,000 units in 100 c.c. of physiologic solution of sodium chloride was started in the form of an intravenous continuous infusion into the same vein. Infusion was continued throughout the experiment at the rate of 34 drops per minute. From the start of the experiment there was no appreciable oozing from the wound, kept moist for a thirty-minute period with a 40 per cent solution of sodium sulfapyridine; at the end of thirty minutes the solution was carefully wiped from the wound. The wound presented an appearance similar to that noted in previous experiments. The wound moistened with physiologic solution of sodium chloride remained moist and continued to ooze throughout the experiment, and for at least thirty minutes after termination of the experiment.

CLINICAL STUDIES

At the outset it was realized that the most difficult aspect of the clinical part of the experiment would be evaluation of results. Controls would not be available. It was therefore necessary to arrive at relatively standard conditions under which an attempt would be made to employ the drug; then an attempt would have to be made to judge the results in a purely uncontrolled and subjective manner. The following criteria were decided upon, in choosing cases for study:

It was decided that, first of all, bleeding must be of the slow, oozing type. It might have occurred at any time from a few hours to fourteen days after tonsillectomy (recurring or secondary hemorrhage). Simple and ordinary methods of control of such hemorrhage, such as thorough cleaning out of the fossa, and the application of mild pressure with a gauze pad for a period of five minutes, must have failed to provide hemostasis, the patient having first received 1/6 to 1/4 gr. (0.01 to 0.016 gm.) of morphine sulfate by hypodermic injection. If these measures had been initiated and had not succeeded, the wound was considered a suitable one for application of the drug. Such standards necessarily meant that only a small group of cases could be had, but it was felt that the standards would

provide far more worthwhile results than those obtained by less rigid standards.

An opportunity for observation of the action of these drugs in the treatment of hemorrhage after tonsillectomy was presented in seven clinical cases, this being the total number of cases judged suitable for consideration during an eighteen-month period at the Worrall Hospital in Rochester. In the first three of these cases, sulfamethylthiazole powder was employed. As previously described, the patient first received a hypodermic injection of from 1/6 to 1/4 gr. (0.01 to 0.016 gm.) of morphine sulfate; after a period of about fifteen minutes the tonsillar fossa was wiped clean after removal of the clot. If bleeding continued after this procedure, pressure was applied in the tonsillar fossa by means of a dry sponge held with a hemostat. This was continued for a period of five minutes. If bleeding still continued, the fossa was again wiped clean, and a thin coating of sulfapyridine or sulfamethylthiazole powder was applied with the aid of a powder blower, directly into the fossa. During this procedure the patient was directed to hold his breath when the powder was being applied, so as to avoid the deleterious effects of coughing.

REPORT OF CASES

CASE 1.—A man, 44 years old, with a year and a half history of diabetes mellitus controlled adequately without insulin, was referred for tonsillectomy because of frequent respiratory infections. Except for the diabetes, the results of physical examination were essentially negative. The blood pressure was 150, systolic, and 90, diastolic, expressed in millimeters of mercury. The patient was admitted to the hospital on April 5, 1940, and tonsillectomy was performed on April 7, at which time infected tonsils of medium size were removed by dissection, and bleeding was controlled by ligatures in each fossa. The immediate postoperative convalescence was uneventful, and the patient was allowed to leave the hospital the next day. On the evening of April 12, however, the patient was readmitted to the hospital, bleeding from the left tonsillar fossa. The clot was removed, and the aforementioned customary procedures carried out. Bleeding continued in the form of persistent oozing from the lower pole of the left tonsillar fossa. Sulfamethylthiazole was then sprayed over the zone of bleeding, the fossa having first been wiped clean. The appearance of an adherent yellow scum was noted on the surface sprayed, and the surface remained dry without further oozing or formation of clot. The patient left the hospital the ensuing day and had no further difficulties.

CASE 2.—A woman, 41 years old, complaining of chronic infectious arthritis, had fibrous, infected tonsillar tags of medium size removed by dissection on May 3, 1940. Results of the preoperative clinical examination had been essentially normal except for a blood pressure of 158, systolic, and 92, diastolic, expressed in millimeters of mercury. Anesthesia was produced by the injection of a 0.1 per cent solution of cocaine. During the operation only moderate bleeding occurred and

this was controlled with ligatures. When the patient left the operating room both fossae were dry.

About three hours postoperatively the patient began to expectorate small clots, and examination revealed oozing from the inferior pole of the left tonsillar fossa. The previously described preliminary procedures did not influence the bleeding. The fossa had been oozing at intervals for more than twelve hours before the powder was used. When we did use the sulfamethylthiazole powder, it was applied thinly over the bleeding area. The result was almost immediate hemostasis; the area covered with the powder was seen to be closely covered by a yellow, firmly adherent scum. Additional bleeding did not occur. The patient remained in the hospital until May 5, and was dismissed from the clinic on May 11, by which time complete separation of the slough had occurred without further incident.

CASE 3.—A man, 24 years old, had a two-year history of recurrent colds and sore throat, during which, he said, he had had a "catch" in his voice. Results of the general examination were essentially negative; the blood pressure was 130, systolic, and 80, diastolic, expressed in millimeters of mercury. The tonsils were large, adherent, and badly infected. They were removed on May 27, 1940, by dissection and snare, and bleeding was controlled by ligatures. The patient was allowed to return to his home two days later, the course during the interim having been without incident. Six days later (June 1) the patient was readmitted, bleeding from the right tonsillar fossa. Bleeding had begun about two hours before his admission and was becoming more profuse. After removal of the clot, it was noted that the bleeding had the form of slow oozing from the entire base of the tonsillar fossa. Sedation and pressure failed to control the bleeding and sulfamethylthiazole was placed in the fossa, with the usual satisfactory result. The patient was kept in the hospital until June 4, when he was dismissed to return to his home. Convalescence was uneventful.

CASE 4.—A man, 54 years old, was admitted to the hospital on June 13, 1940. He was known to have had moderately severe diabetes of four years' duration, and at the time of admission required 25 units of protamine-zinc insulin daily. Three months previously he had had acute tonsillitis. The blood pressure was 140, systolic, and 78, diastolic, expressed in millimeters of mercury; otherwise results of the physical examination were not remarkable.

On June 19 tonsillectomy was performed, and very large, septic tonsils were removed by dissection, from fossae to which they were densely adherent. Bleeding at multiple points was controlled with catgut ligatures. The immediate postoperative course was uneventful but the day after tonsillectomy the patient began to spit small quantities of blood. General oozing from both fossae was discovered. Sedative agents and pressure did not control the bleeding, and treatment of the fossae with sulfapyridine was instituted. The fossae were first wiped dry; then a thin spray of powdered sulfapyridine was employed. The effect was immediate. Bleeding was definitely controlled, and the fossae appeared dry and coated by the powder. No further difficulty was encountered, and the patient was dismissed from the hospital on June 21 and from the clinic on June 26.

CASE 5.—A man, 37 years old, who complained chiefly of severe generalized chronic fibrositis, entered the hospital June 26, 1940, for the purpose of undergoing tonsillectomy the next day. Blood pressure was 122, systolic, and 84, diastolic,

expressed in millimeters of mercury. Results of the physical examination otherwise were not significant. At operation large septic tonsils were dissected out and profuse bleeding was controlled by ligatures placed in both fossae. There was no bleeding when the patient left the operating room. During the day he spat a few small clots, but there was no real bleeding. About thirty-two hours postoperatively, however, bleeding from the left tonsillar fossa occurred, from a diffuse area situated at the base of the fossa. Sedative agents, removal of clots and pressure were used without results. Sulfapyridine was then applied in the usual manner and the oozing stopped almost instantaneously. The previously bleeding area was seen to be dry and coated with a film of hardened powder. No further difficulties were encountered, and the patient left the hospital on June 29, and was dismissed from the clinic on July 5.

CASE 6.—A woman, 59 years old, who had chronic infectious arthritis, had had episodes of sore throat and tonsillitis for thirteen years prior to her admission to the clinic. Blood pressure was 210, systolic, and 124, diastolic, expressed in millimeters of mercury. She was admitted to the hospital for tonsillectomy, which was performed on July 18, 1940. At that time large septic tonsils were removed by dissection and snare. Minimal bleeding occurred, and was easily controlled with ligatures in both fossae.

The postoperative course was uneventful, and on July 20 the patient was dismissed from the hospital. On the evening of July 23, without any apparent precipitating factor, the patient began to spit blood. Profuse bleeding from the left tonsillar fossa was noted. The clot was cleaned from the fossa, and 1/4 gr. (0.016 gm.) of morphine sulfate was administered hypodermically. Pressure was then applied, but oozing continued from a large area at the base of the fossa. Sulfapyridine powder next was sprayed over the bleeding area. The result was drying of the fossa and caking of the sprayed powder over the surface of the wound. No further bleeding was encountered, and the patient was dismissed from the hospital three days later, and from the clinic on July 29.

CASE 7.—A woman, 50 years old, suffered from severe chronic infectious arthritis. Blood pressure was 130, systolic, and 90, diastolic, expressed in millimeters of mercury. Results of the physical examination otherwise were essentially negative. The patient was admitted to the hospital on May 30, 1941, for tonsillectomy on the next day. At operation, large, infected tonsils were removed by dissection and snare. Considerable bleeding was encountered on the right side, and was controlled by ligatures. The postoperative course was uneventful except for pain in the right ear which extended in the direction of the eustachian tube. This pain arose on the day after operation and remained for more than two days. She was dismissed from the hospital on June 3. On the night of June 6, the patient was readmitted to the hospital, bleeding from the right tonsillar fossa. Sedative agents and the usual cleaning of the fossa and the application of pressure were without lasting results, and oozing from the area continued at intervals for about eighteen hours. At that time the fossa was cleaned of clot and a thin coating of sulfapyridine powder was applied to the bleeding area. Immediate hemostasis was noted and the area was as usual covered with a thin, hard, white crust. No further bleeding occurred and the patient was dismissed from the hospital on June 8, and from the clinic on June 11.

COMMENT

As we have previously noted, it is extremely difficult to conduct a clinical study of this nature in anything like a scientific manner. There is no way of adequately controlling the study. It therefore seemed that the best that could be done would be to adhere to conditions of a standard nature under which the drugs could be applied to the bleeding surface; and it seemed desirable also that in each case the bleeding area should first have been treated by methods other than ours and methods which were the same in all cases.

Thus, the only patients we treated were those who had not responded to measures usually sufficient to control hemorrhage, and measures which are routine procedures in such cases. Hence, if the drug was of value in these cases, it surely should serve in situations less severe. To assure similarity in the method of application of the drug in each case and to avoid unnecessary trauma to the tissues, the powdered drug was applied by spraying from a powder blower,* and an amount just sufficient to cover thoroughly the bleeding area was used. Observations were made immediately. Patients were observed at one- to two-day intervals during their convalescence, but nothing significant was noted other than the immediate reaction, as described herein.

An attempt was made experimentally to produce granulation tissue in the laboratory which would resemble such tissue as found in the tonsillar fossa at the time most secondary bleeding occurs. No attempt was made to use the mucous membranes of animals, and the fact that the experimental wounds thus were dry, rather than moist, was not considered to be significant, since the reaction which occurred was immediate, and hence would not be influenced by environment to any appreciable extent.

The results of clinical and experimental studies combined are such that it can be said definitely, we believe, that sulfapyridine and sulfamethylthiazole powders applied locally to an area of oozing or capillary bleeding have an immediate hemostatic effect.

The mechanism by which this is accomplished is not known. We do know that small, unbroken blood vessels are not affected by the drugs in question, whereas open and bleeding vessels immediately disappear from view. Large numbers of erythrocytes are noted in sections made after treatment of wounds with these drugs.

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*De Vilbiss Co., Toledo, Ohio.

The suggestion that the effect might be caused simply by the physical properties of the substances used is negated by the fact that sulfanilamide and sulfathiazole powders and talcum powder similarly applied had little or no effect. Strong solutions of sodium sulfapyridine also have a hemostatic effect; this fact would seem to be unrelated to the high degree of alkalinity of the compound, since a solution of sodium hydroxide of the same hydrogen ion concentration had no effect on bleeding. Results obtained in work in the experimental laboratory and therapeutically for patients were comparable.

On the bronchoscopic service we have recently used sulfapyridine powder by insufflation in the control of persistent bleeding in a case of bronchial erosion. Although the time-interval since treatment in this case has not been great, there has as yet (the time of this report) been no recurrence of hemoptysis except for a small amount of blood-streaked sputum which endured for two days after the treatment.

SUMMARY AND CONCLUSIONS

The application of powdered sulfapyridine and sulfamethylthiazole to experimental wounds in the horse, guinea pig and rabbit result uniformly in the control of vascular oozing. These results confirm observations made in cases of delayed and secondary tonsillar hemorrhage in which the aforementioned drugs were used successfully in the control of persistent bleeding.

A 40 per cent solution of sodium sulfapyridine was found to be equally efficacious experimentally, and its use has made possible detailed observation of the process of control of bleeding by this method. The exact mechanism at work is open to conjecture, however.

Sulfanilamide and sulfathiazole powders proved to have but little hemostatic value. Talc used as a control was of no value. A solution of sodium hydroxide with a hydrogen ion concentration closely simulating that of sodium sulfapyridine failed to produce comparable results when it was used experimentally.

The finely powdered drugs were found to be easy to apply if they were kept dry and were insufflated directly onto the surface of the wound by the use of a small powder blower. The sulfamido compounds used obviously were bacteriostatic, and perhaps bactericidal.

It would seem, on the basis of clinical and experimental results, that sulfapyridine and sulfamethylthiazole powders may be considered to be simple, safe and effective therapeutic agents for local application to the tonsillar fossa in the control of secondary tonsillar hemorrhage not amenable to routine management.

These observations have added one more use to the long list of uses for the chemotherapeutic drugs; that of control of slow oozing hemorrhage through local application.

The sulfamido drugs are at least bacteriostatic, perhaps bactericidal, and if infection has been a factor in the causation of hemorrhage, as many authors suggest, their application in the control of bleeding should help to suppress infection in the wound and thus serve a double purpose.

It is possible that the routine use of sulfapyridine postoperatively in the tonsillar fossa might tend to prevent the occurrence of secondary hemorrhage. Studies in this respect have already been started, but a considerable number of cases will be necessary before any conclusions can be drawn from the work.

The drugs should also be of value when locally applied to superficial abrasions such as brush burns.

THE MAYO CLINIC.

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XXX

THE DEVELOPMENT OF THE OLFACTORY NERVE, THE NERVUS TERMINALIS, AND THE VOMERONASAL NERVE IN MAN*†

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The olfactory nerve, the nervus terminalis and the vomeronasal nerve are so closely related that it is difficult to describe the development of one of them without a consideration of the other two nerves. Until the finer structure of the olfactory nerve was demonstrated by the use of methylene blue and the Golgi method in the latter part of the nineteenth century, the olfactory nerve was poorly understood. Confusion existed as to whether the olfactory nerve fibers were outgrowths from the brain, were developed from the neural crests, or grew into the brain from the membranous lining of the nose. All three nerves have their origin in the olfactory placode and they grow into the brain. His[1] was one of the first to point out that the olfactory nerve grows in from a peripheral thickening of the ectoderm, but he failed to recognize the presence of three nerves.

This study is part of a research program on the development of the cranial nerves in man. The author's material was supplemented through the use of the embryological collections of Professor W. F. Windle, of Northwestern University, and of Professor G. W. Bartelmez, of the University of Chicago. The literature on this subject has been discussed in recent papers by the author[2, 3] and for this reason will receive only brief consideration here. The following will be a brief summary of the observations on the development of the nerves in the nose.

A description of the development of these three nerves would naturally begin with a consideration of the olfactory placode. The olfactory placodes can be first recognized as slight thickenings of the ectoderm at the rostral end of the embryo. In young human embryos of about twenty-six somites, these thickenings of the ectoderm occur

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†Read before the Chicago Laryngological and Otological Society, Nov. 3, 1941.

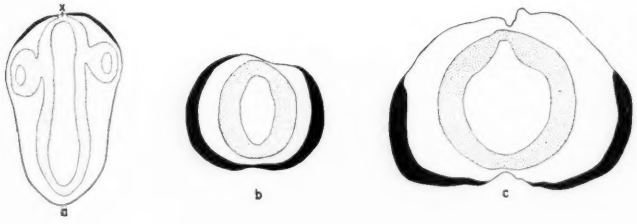


Fig. 1.—Sections through the forebrain (stippled) and olfactory placodes (solid black) of young human embryos. *a*. Horizontal section of a 4-week embryo. *x* indicates anterior neuropore. *b*. Transverse section of a 4-week embryo. *c*. Transverse section of a 4½-week embryo.

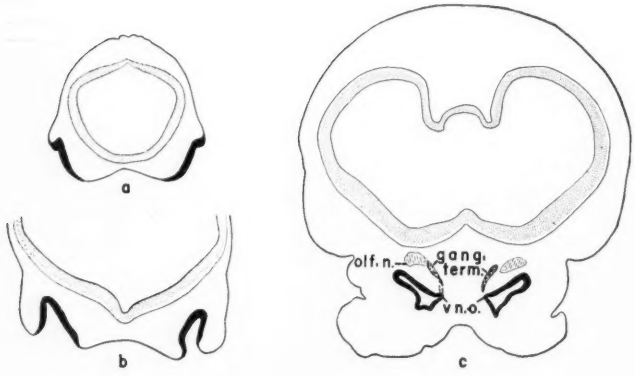


Fig. 2.—Transverse sections through the head region of young human embryos. The forebrain is stippled and the olfactory placodes and the lining of the nasal fossae are black. *a*. 5-week embryo. *b*. 5½-week embryo. *c*. 6-week embryo. *vn.o.* Vomeronasal organ.

on either side of the closed anterior neuropore (Fig. 1*a*). The epithelium of the placode changes from a low type of epithelium to tall columnar epithelium and then to pseudostratified columnar epithelium. The olfactory placodes are at first poorly delimited from the adjoining ectoderm. As the development continues, the olfactory placodes become more clearly defined and, because of differential growth, change from a lateral to a ventrolateral position (Figs. 1*b* and 1*c*). The general plan of how the olfactory placode invaginates to form the lining of the nasal fossa (Fig. 2) and how the vomeronasal organ (of Jacobson) is formed by an evagination or an outgrowth of the medial wall of the nasal sac is well told in the standard textbooks of embryology and needs no further consideration here.

Bipolar neuroblasts have begun to develop in the olfactory epithelium of human embryos of about 8 or 9 mm. The central processes of these cells grow toward the brain. As these nerve fibers approach the brain, they are accompanied by cells which have wandered out from the olfactory epithelium. These fibers and cells form small bundles and cords which extend toward the forebrain. At first these cords are attached only to the olfactory epithelium, but later they become connected to the brain also. With continued growth the small bundles unite to form a large bundle which stretches obliquely upward from the olfactory epithelium to the ventral wall of the forebrain. This bundle is the embryonic olfactory nerve (Fig. 3*a*). The olfactory nerve fibers have their cells of origin in the epithelium of the nasal fossa just lateral to the anlage of the vomeronasal organ. Many of the elements which form the ganglion terminale and the vomeronasal nerve arise from the anlage of the vomeronasal organ and are located along the medial side of the olfactory nerve (Fig. 2*c*). The olfactory nerve fibers are not easily stained in young embryos, and it is difficult to determine just when the fibers begin to grow into the brain. These fibers have probably begun to enter the brain by the sixth week of human development.

When the olfactory nerve first contacts the brain, there is no indication of an olfactory bulb. It is only after the fibers of the olfactory nerve have begun to grow into the brain that the olfactory bulb begins to show signs of development. A slight bulge on the outside of the forebrain where the olfactory nerve is attached and a small depression on the inside of the brain are the first indications that the olfactory bulb has begun to develop (Fig. 3*b*). The olfactory bulb begins to show signs of development in about the seventh week of human development.

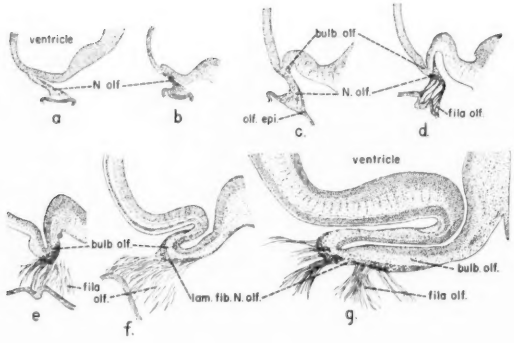


Fig. 3.—The development of the olfactory bulb is illustrated in the sagittal sections of human embryos and fetuses. The following stages are shown: *a*. 7 weeks. *b*. 7 weeks (slightly older than *a*). *c*. 7½ weeks. *d*. 8½ weeks. *e*. 9 weeks. *f*. 11 weeks. *g*. 13 weeks. (From the *Journal of Comparative Neurology*, Wistar Institute of Anatomy.)

With the additional growth of olfactory nerve fibers toward the brain, there is a gradual increase in the size of the olfactory nerve. The central end of the nerve is attached to the developing olfactory bulb (Fig. 3*c*). The olfactory bulb develops as an outgrowth of the wall of the forebrain. The ventricle of the brain extends into this outgrowth or evagination. In human embryos of about 22 mm. (Fig. 4), the olfactory bulb is seen as a distinct protrusion of the brain which is directed downward, backward, and slightly medialward. The proximal end of the olfactory nerve fits snugly over the end of the developing olfactory bulb.

In these young embryos the elements which form the nervus terminalis and the vomeronasal nerve lie along the caudomedial side of the olfactory nerve (Figs. 5 and 6). The attachment of these nerves to the brain is just caudal to the attachment of the olfactory nerve. The relationship is very close but an irregular boundary can be seen between them. The larger cells of the ganglion terminale stand out in contrast to the small cells among the fibers of the olfactory nerve. Sheath cells are thought to develop from small cells among the fibers of the olfactory nerve.

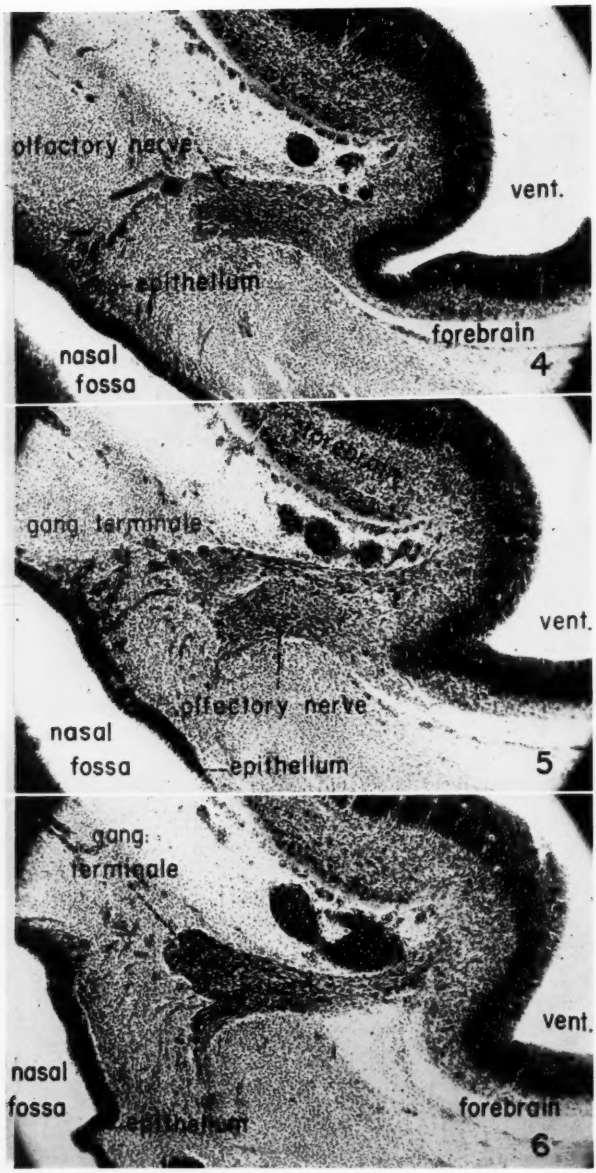
The olfactory bulb continues to enlarge. During the eighth and ninth weeks the bulb is gradually directed downward and medial-

ward (Figs. 3*d* and 3*e*). A slight constriction develops at the base of the developing bulb which indicates a boundary between the bulb proper and the rest of the brain. The large bundle of fibers which constitutes the embryonic olfactory nerve gradually becomes part of the olfactory bulb. The embryonic olfactory nerve spreads out into a loose plexus of fibers and cells (sheath) which partially covers the surface of the olfactory bulb. This layer is known as the layer of olfactory nerve fibers. As the nerve fibers arising in the olfactory epithelium increase in number, they are grouped in many small bundles and these are known as the fila olfactoria. The fila olfactoria pass without interruption into the layer of olfactory nerve fibers of the bulb.

During development there is a gradual rotation in the position of the olfactory bulb. At first it is directed downward, backward, and slightly medialward. Through growth changes it is gradually directed downward and medialward. During the tenth and eleventh weeks of human development the bulb is gradually directed forward (Figs. 3*f* and 7). After the olfactory bulb is directed forward, it begins to grow more in length than in thickness (Fig. 3*g*). This is well shown in fetuses of thirteen weeks and older. The fila olfactoria are collected into fewer but larger bundles with the formation of the cribriform plate. The increase in the number of olfactory nerve fibers from the olfactory epithelium is evidenced by the size of the fila olfactoria and the increased thickness of the layer of olfactory nerve fibers of the bulb. Secondary olfactory connections are in the process of being established within the brain by the beginning of the third month of fetal development. These connections are beyond the scope of this paper.

The migration of cells from the olfactory epithelium along the olfactory nerve fibers begins early and continues over a considerable period of development. Clusters and cords of these cells are shown along the slender filaments of the fila olfactoria of an eleven-week fetus (Fig. 8). These cells which migrate along the fibers of the fila olfactoria are thought to develop into sheath cells. The ventricle of the brain continues out into the olfactory bulb until about the fourteenth week of human development. Humphrey[1] has shown that cells from the ependyma begin to grow into the olfactory ventricle about this time, and by the nineteenth week the olfactory ventricle has been obliterated.

In Kipling's story, "The Elephant's Child", there is a struggle between a crocodile and an elephant. During the struggle the crocodile pulls the elephant's nose out into a long snout. The de-


olfactory nerve
vent.
epithelium
forebrain
nasal fossa
4
forebrain
gang. terminale
vent.
olfactory nerve
nasal fossa
epithelium
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gang. terminale
vent.
nasal fossa
epithelium
forebrain
6

Fig. 4.—A sagittal section through the developing olfactory bulb and the olfactory nerve of a 7½-week human embryo. Protargol preparation.

Fig. 5.—The close relation of the olfactory nerve and the ganglion terminale is shown in a sagittal section of the same embryo shown above. The plane of the section is slightly medial to that of Fig. 4. Protargol preparation.

Fig. 6.—Another sagittal section of the same embryo. The plane of the section is slightly medial to Fig. 5. Note the relation of ganglion terminale. Protargol preparation.

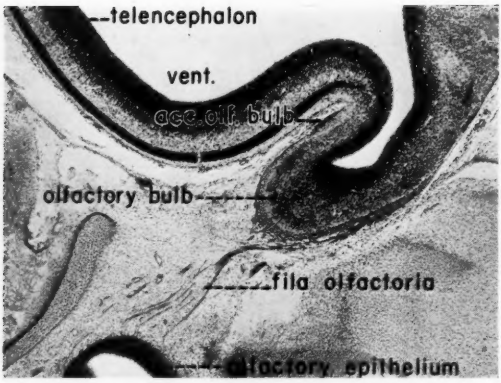

Fig. 7.—A sagittal section through the olfactory bulb of a 11-week human fetus. Protargol preparation.

velopment of the olfactory bulb reminds one of this story. The olfactory nerve would correspond to the crocodile and the brain to the elephant. The olfactory nerve does not actually pull part of the brain out into a prolongation; however, it does exert a great influence on the development of the olfactory bulb. The experimental work of Burr[5] and others on amphibians suggests that a reciprocal relation exists between the olfactory nerve and the olfactory region of the brain. The rapidly multiplying cells of the brain attract the growing olfactory nerve fibers like a magnet and cause these fibers to enter the brain. The entrance of the olfactory nerve fibers into the brain appears to stimulate the cells in that region to multiply more rapidly than before. A hyperplasia results which is thought to play a direct role in the formation of the olfactory bulb. The degree of development of the olfactory bulb is probably in proportion to the number of olfactory nerve fibers which enter the brain. The stimulus associated with the ingrowing nerve fibers is thought to be the main cause of the hyperplasia.

Although the nervus terminalis was recognized as a distinct nerve only in the latter part of the nineteenth century, it was soon identified in all classes of vertebrates, with the possible exception of birds. The nerve was first described in a fish by Pinkus (1895), and for a time it was called the nerve of Pinkus. Professor Locy (1905), of Northwestern University, suggested the name "nervus terminalis", and this term has been generally adopted. Perhaps the

best description of the nervus terminalis in a mammal was made by Huber and Guild,[6] based on young rabbit preparation. In the rabbit the nervus terminalis is an independent nerve whose peripheral course is through a loose plexus. Ganglion cells are scattered along the course of the nerve. The largest group of these ganglion cells is known as the ganglion terminale. Part of the fibers of the nervus terminalis follow the peripheral path of the vomeronasal nerve, while others distribute to the septal mucous membrane in front of the vomeronasal nerve. From the dissections of the human fetus and infant, McCotter[7] found the peripheral course of this nerve similar to that described for the rabbit. In adult man the course of the nervus terminalis within the cranium has been found to lie over the surface of the gyrus rectus, and the entrance of the nerve into the brain to be in the region of the medial olfactory striae.[8]

The nervus terminalis is more intimately related to the development of the vomeronasal nerve than to the olfactory nerve. Unless differential staining is used, the two nerves can scarcely be distinguished. The ganglion terminale and the vomeronasal nerve arise from the region of the anlage of the vomeronasal organ (Figs. 2*c* and 9). This is located in the medial wall of the olfactory fossa. From this region cells and fibers migrate to a position along the medial side of the embryonic olfactory nerve. Irregular masses of cells and fibers are formed which constitute the ganglion terminale. In young embryos it is not possible to distinguish clearly between the elements of the two nerves. The olfactory nerve fibers are distinct because they arise in the part of the olfactory placode lateral to the anlage of the vomeronasal organ.

The migration of cells and fibers continues in the formation of the ganglion terminale during a considerable period of growth. One end of the ganglion becomes connected to the forebrain. The fibers from the ganglion continue into the wall of the forebrain. The cells among the fibers form a continuous stream from the forebrain out into the ganglion. The arrangement of the cells suggests that there may be a migration of cells from the brain out into the ganglion. The distal end of the ganglion breaks up into a number of branches. The ganglion terminale of a 22 mm. human embryo lies close along the medial border of the olfactory nerve (Figs. 5 and 6). In places the two structures look almost continuous. A boundary, however, can be distinguished. During development the ganglion terminale begins to separate from the embryonic olfactory nerve and from the forebrain. The ganglion, however, retains a connection with the forebrain by fiber bundles (Fig. 10).

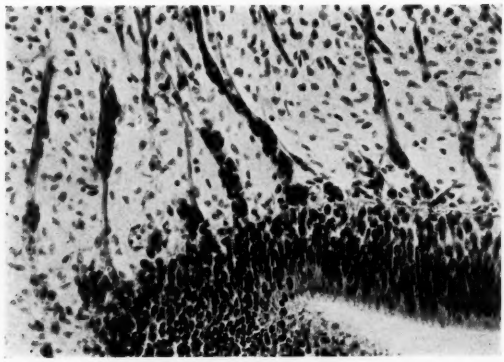

Fig. 8.—A sagittal section through the fila olfactoria and the olfactory epithelium of a 11-week fetus. Note the migratory cells along the olfactory nerve fibers. Protargol preparation.

As the olfactory bulb begins to take form, the ganglion terminale is located along its medial border. The branches of the nervus terminalis which distribute to the anterior septal region enter the rostral end of the ganglion terminale. The vomeronasal nerve and the branches of the nervus terminalis which accompany this nerve enter the ventral border of the ganglion terminale. The central roots of the nervus terminalis collect at the caudal border of the ganglion and extend to the base of the olfactory bulb where they enter the forebrain. The ganglion terminale consists of a network of interlacing fibers with many ganglion cells among the fibers.

It is not always possible to distinguish the types of ganglion cells among the fibers of the nervus terminalis. Unipolar, bipolar and multipolar cells were observed. Some authors have claimed that the multipolar cells resemble sympathetic ganglion cells. Brookover[9] reported the presence of over 1500 ganglion cells along the peripheral course of the nervus terminalis in addition to the many nerve cells in the ganglion terminale.

The details of the central course of the nervus terminalis are not easily followed in young human embryos. Fibers from the ganglion terminale can be followed caudad into the forebrain but they are soon lost. In older embryos and in fetuses the central roots of the nervus terminalis are more distinct and can be followed caudad from the ganglion terminale. Their place of entrance into the forebrain is just caudal to the olfactory bulb. These fibers sink deep into

the brain. Some of them can be followed into the septal area, and others extend in a more caudal direction toward the hypothalamus. They soon disappear and their ultimate termination is not known. The fibers which reach the septal area of the brain appear to end in the medial and lateral septal nuclei. The central roots of the nervus terminalis are longer in older fetuses. This is to be expected because the ganglion terminale is farther removed from the place of the attachment of its roots to the forebrain.

The fibers of the nervus terminalis are sometimes differentially stained in pyridine silver preparation. In such preparations, the olfactory and the vomeronasal nerves may be lightly stained while the fibers of the nervus terminalis are darkly stained. It is then possible to follow the main course of the nervus terminalis. Several branches of the nervus terminalis leave the ventral border of the ganglion terminale and follow the vomeronasal nerve through the cribriform plate. The dark fibers of the nervus terminalis are easily distinguishable from the more lightly stained fibers of the vomeronasal nerve. The dark fibers of the nervus terminalis can be traced toward the vomeronasal organ. It is difficult to tell their exact termination because they gradually disappear as the vomeronasal organ is approached. The path of these branches through the cribriform plate and through the mucous membrane of the nasal septum is usually deep (medial) to the fila olfactoria.

A division of the nervus terminalis from the rostral end of the ganglion terminale distributes to the anterior region of the nasal septum (Fig. 11). These fibers spread out into a loose plexus of fibers deep to the fila olfactoria. This plexus or network is made of fine fibers with cells scattered among the fibers. It has reminded some authors[6] of the enteric plexus. The termination of these fibers has been difficult to ascertain. Some of these fibers may distribute to the glands in the nasal septum and others may terminate in the epithelium. Cells migrate from the epithelium of the rostral border of the septal mucous membrane along the fibers of the anterior septal branch of the nervus terminalis. A chain of these migratory cells is shown in Fig. 12.

The anterior septal branch of the nervus terminalis is usually joined by the medial nasal branch of the anterior ethmoid nerve (of V). These two nerve bundles run together for some distance. Their ultimate termination is not shown in this material.

It is quite likely that there are sensory and autonomic components within the nervus terminalis. The bipolar and the unipolar

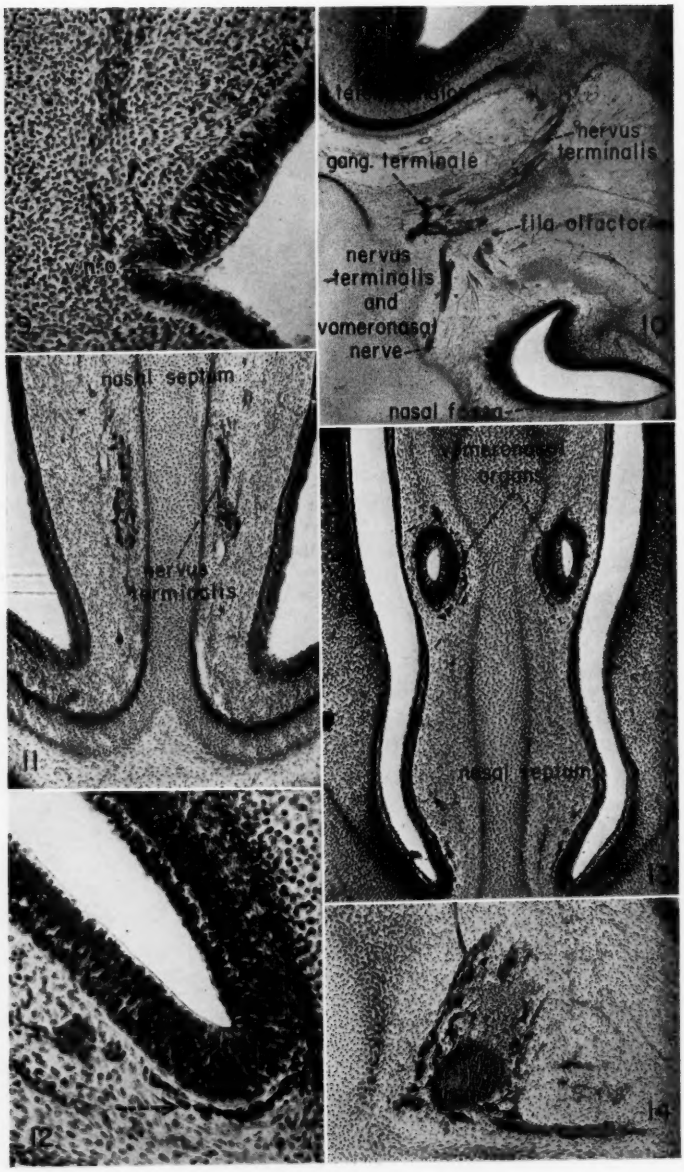
gang. terminale
nervus terminalis
fila olfactor
nervus terminalis and vomeronasal nerve
10
nasal septum
nervus terminalis
11
vomeronasal organs
nasal septum
13
12
14

Fig. 9.—A transverse section through the medial wall of the olfactory fossa of a 6-week human embryo. Note the fibers and cells of the nervus terminalis and vomeronasal nerve which have migrated from the anlage of the vomeronasal organ *(vn.o.)*. Protargol preparation.

Fig. 10.—A sagittal section through the forebrain and the ganglion terminale of a 11-week human fetus. The plane of the section is medial to that of Fig. 7. Protargol preparation.

Fig. 11.—A transverse section through the nasal septum of a 9-week human fetus. Note the anterior septal branch of the nervus terminalis. Protargol preparation.

Fig. 12.—A transverse section through the rostral part of the nasal mucous membrane of a 9-week human fetus. Note the chain of migratory cells indicated by the arrow. Protargol preparation.

Fig. 13.—A transverse section through the nasal septum of a 9-week human fetus. Note the vomeronasal organs. Protargol preparation.

Fig. 14.—A sagittal section through the vomeronasal organ of a 11-week human fetus. Note the migratory cells. Protargol preparation.

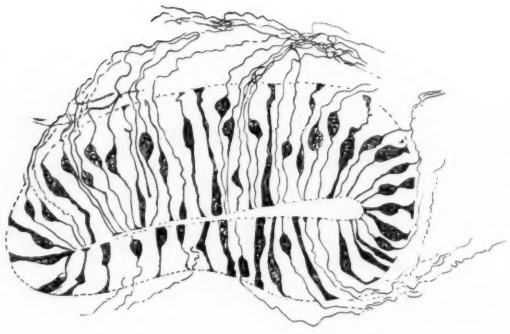

Fig. 15.—Cross-section of the vomeronasal organ of a rabbit fetus. Golgi method. (Redrawn from von Lenhossek.[12])

neurons probably make up the sensory part, while the multipolar neurons probably belong to the autonomic part. Embryonic material does not give a complete picture of the distribution of the fibers of the nervus terminalis, as there are many glands in the nasal septum which form during late fetal development. The nervus terminalis may be related to these glands. Although this nerve has been described in nearly every class of vertebrates, its function is still unknown. There is still much to be learned about this most interesting nerve.

The general structure and development of the vomeronasal (Jacobson's) organ (Fig. 13) is well known. It will be sufficient for this discussion to point out that the organ develops as a sac-like outgrowth from the medial wall of the nasal fossa (Figs. 2*c* and 9). It is said to reach its height of development during fetal life, after which the organ usually degenerates. The organ contains neuroepithelium in which there are nerve cells resembling typical olfactory cells (Fig. 15). The vomeronasal nerve is composed of fibers which are the processes of these nerve cells. The fibers of this nerve are gathered into several bundles which branch and anastomose and finally converge in the ganglion terminale.

In certain lower mammals (the rabbit and the opossum) the vomeronasal nerve enters an area of the brain which has been designated the accessory olfactory bulb.[10] This area is located along the dorsomedial side of the olfactory bulb, and such an area can be seen in human fetuses (Fig. 7). It is not possible, however, to demon-

strate a large root of the vomeronasal nerve to the accessory olfactory bulb in human fetuses such as has been described in the rabbit and the opossum. A number of small bundles leave the vomeronasal and nervus terminalis complex and enter the medial side of the olfactory bulb in front of and along the lower border of the accessory olfactory bulb. Comparative anatomical studies have shown that the size of the accessory olfactory bulb varies directly with the size of the vomeronasal organ. Since the vomeronasal organ is considered to be rudimentary in man, it is not surprising that the central roots of the vomeronasal nerve are inconspicuous. The accessory olfactory bulb probably disappears with the degeneration of the vomeronasal organ. The accessory olfactory bulb is reported to be lacking in the adult human brain. An area denuded of olfactory formation was found in its place.[11]

It is interesting to point out the great proliferation of cells from the developing vomeronasal organ (Fig. 14). These cells migrate along the fibers of the vomeronasal nerve, along the nervus terminalis, and along a branch of the nasopalatine nerve which runs to the vomeronasal organ. These migratory cells are thought to develop into sheath cells and ganglion cells.

SUMMARY

A distinct olfactory nerve exists in the development of the human embryo. During the development the main mass of this nerve becomes incorporated as a layer of the olfactory bulb. The olfactory bulb begins to develop after the attachment of the olfactory nerve to the brain. The bulb then forms as an outgrowth of the brain. The bulb undergoes a rotation in position during its development. Fig. 3 summarizes the development of the olfactory nerve and the olfactory bulb.

The nervus terminalis and the ganglion terminale are formed by the growth of fibers and the migration of cells from the region of the anlage of the vomeronasal organ. These elements coalesce along the medial side of the embryonic olfactory nerve. One end of the ganglion becomes attached to the ventromedial wall of the forebrain. During development the ganglion becomes separated from the brain, but its connection with the brain is retained by fiber bundles. These fiber bundles constitute the central roots of the nervus terminalis and enter the brain just caudal to the olfactory bulb. The central roots of the nervus terminalis sink deep into the brain and some of their fibers reach the septal nuclei. The peripheral branches of the nervus terminalis distribute to the mucous

membrane of the nasal septum. Part of these fibers follow the course of the vomeronasal nerve, while others distribute to the region in front of this nerve.

An accessory olfactory bulb comparable to that described in lower mammals is present in the human fetus. This area of the brain receives the fibers of the vomeronasal nerve.

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XXXI

OTOLOGY AND AVIATION*†

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The medical research board established by the army through Colonels Lyster and Eugene Lewis early in 1917, for investigation of all physical and mental factors pertaining to military aviation, was headed by Prof. Yandell Henderson, with the active collaboration of the late John B. Rae. They established the medical research laboratory at Mineola, which continued until 1920, when a school for flight surgeons was created at Mitchell Field. In 1926 this institution, known since 1922 as the School of Aviation Medicine, was moved to Brooks Field and in 1931 to Randolph Field, Texas. The first commandant of the air medical school, Col. Louis H. Bauer, served from 1919 to 1925. A research laboratory was established in 1933 at Wright Field, Ohio, for intensive study of the effects of flight on the human organism.

Early emphasis on labyrinthine function came to be disregarded by these early observers and teachers of aviation medicine. The flight surgeon—not the otologist—confronted daily with new problems in the examination and conditioning of flying cadets, was obliged to set up for himself as a result of practical experience such methods as he found most reliable. Vision and eye-muscle balance, depth perception, and the neuropsychic, cardiovascular and respiratory mechanisms, all received important revaluation in the light of experimental evidence from the laboratory, the ground school, and eventually from flight conditions which have lately reached the stratosphere.

The auditory apparatus has, from the standpoint of hearing alone, not attracted much more attention from the flight surgeon than has the vestibular apparatus. It has long been accepted that continued exposure to airplane noise would, as with other noisy occupations, lead to fatigue and to upper tone losses. Over-

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†Read before the American Otological Society, Atlantic City, N. J., May 28, 1942.

excitable labyrinths have been found, in sailors and airmen alike, to lose irritability with habituation; and it was found imperative for the "blind" flyer, working from instruments, to disregard the lagging impulses from his canals and to follow his instruments absolutely. Otherwise he might be flying wing down or upside down and not be aware of it.

Such facts, and many more from experience little known to the generality of otologists, led to the first symposium on aviation medicine in 1931, before the American Academy of Ophthalmology and Otolaryngology, and to its subsequent study courses. Within the past few years, papers on otorhinologic subjects related to flight have come before this and other special societies. It has become evident that practical considerations arising from actual flight, checked by repeated laboratory work in reduced pressure chambers, have determined present standards for army and navy pilots, and in large measure for commercial airline pilots, many of whom hold reserve commissions.

Elementary as they may seem, it may prove interesting to review these military physical standards, and to discuss therewith the reactions of civilian passengers subjected to varying conditions during flight.

Applicants for flying training are rejected if any abnormality of either ear be present. The external auditory canal must be open, free from eczema, boils or fungus infection, not obstructed by exostoses or unduly small in diameter. The tympanic membrane must have normal luster and inclination as evidenced by the light reflex. Any inflammation, perforation, or thin, atrophic scarred area, with or without history of previous otitis, disqualifies. Small, firmly healed scars not impeding normal movement of the membrane do not disqualify. Retraction of the tympanic membrane which recurs or cannot be corrected by inflation disqualifies. History and local evidence of a radical mastoidectomy, even if hearing and balance are normal, disqualify. A simple mastoidectomy, well healed without other abnormality, does not disqualify. History of otitis media without recurrence for six months, and without previous attacks, will not disqualify if hearing and other tests are normal; but repeated acute exacerbations require rejection. Persistent or recurrent tinnitus for more than six months also disqualifies, particularly if associated with local pathologic findings.

Hearing is tested by whispering numerals, including 66, 18, and 23, from a distance of 20 feet, using residual air in the lungs

at the end of an expiration. The applicant faces away from the examiner, plugging first the left, then the right meatus with his finger. Care must be taken to see that complete closure is made. On original examination, 20/20 is required for each ear; expert pilots on re-examination, observers and other nonpiloting personnel of the air force are permitted 8/20 in each ear, or 15/20 in one and 5/20 in the other. Audiometric examination is made and recorded annually where such instruments are available; the average hearing loss permissible for pilots on original examination must not exceed 15 per cent.

Patency of the eustachian tube is tested by the Politzer bag if the tympanic membranes are retracted and the light reflex slants abnormally. During inflation the membrane should be observed to note change in its reflex; in doubtful cases an auscultation tube to the examiner's ear will permit the inrush of air to be heard. Obviously, any obstructive lesion of the nose or pharynx—adenoids, aberrant lymphoid tissue, excessively large tonsils, septal thickening or deviation, hypertrophied or allergic turbinates, nasal polypi or chronic sinusitis—may interfere with normal opening of the eustachian tube during swallowing, and thus with the aeration of the middle ear. Blocking of the eustachian tube disqualifies all classes of applicants.

Tests of equilibrium have been greatly simplified, except where a history of instability—train sickness, seasickness, airsickness—has been elicited. Ordinarily, the self-balancing test is adequate; the applicant stands erect without shoes, heels and toes touching. He then flexes one knee backward to a right angle without bending the hip, thus avoiding support against the other leg, closes his eyes, and holds the position if possible for 15 seconds. The test is repeated on the other foot. No hopping or moving about is permitted. The bended leg may be moved back and forth, or the arms moved, to assist balance. Findings are recorded as "steady" when no appreciable movement occurs; "fairly steady" when the bended leg or arms are moved slightly; "unsteady" when he manifestly has much difficulty in keeping his balance; and "failed" when he cannot hold his position for 15 seconds, one out of three trials on each foot.

The vestibular test with a turning chair is made on original examination, when there is unsteadiness on the self-balancing test, or marked tremor of eyelids and fingers, unsteady Romberg and gait, or sitting pulse rate above 90; and especially when a history of sickness or other vertigo associated with motion has been brought out. The applicant is seated with the head 30 degrees forward,

tragus of ear level with external canthus of eye. He is then required to fix a distant point with his eyes and the chair is turned slowly from side to side to note whether spontaneous nystagmus is present. The eyes are then closed, and the chair is turned to the right, ten turns in 20 seconds; chair is stopped, eyes opened and fixed on a distant point; horizontal nystagmus to the left for 26 seconds should occur. Conversely, the applicant next being turned to the left in the same manner, nystagmus to the right occurs. Nystagmus of 10 to 34 seconds, if approximately alike in the two directions, is permissible. Any variation of more than 5 seconds between the two directions disqualifies. Further evidence of instability is detected by taking pulse and blood pressure before and after rotation; marked increase in pulse and systolic pressure (30 or more points); marked fall in diastolic pressure (10 points or more).

While the sense of equilibrium is not wholly dependent upon the vestibular apparatus, its normal reactions to change of posture are a necessary part of the pilot's ability to perceive his position in space. Somatic sensations, deep muscle sense and particularly the corrective value of well-balanced vision and depth perception all contribute to correct and modify impulses received from the semicircular canals and the otolith apparatus. Turning chair tests are of course valuable adjuncts to the self-balancing procedure, but the latter has been found adequate for exclusion of almost all individuals with sensitive labyrinthine reactions. Inequalities between the two sides in reaction time are suggestve of an unrecognized lesion of the middle ear, perhaps with crusts or cholesteatoma concealed in a high perforation; or, more rarely, of circulatory or neoplastic disease somewhere along the course of the vestibular nerve fibers, either in the internal auditory meatus or at the cerebellopontile angle. The caloric test, since it brings about vestibular stimulation of one ear at a time, is valued for differential diagnosis in civil practice, but is not ordinarily used in military examinations. Douching of the ear by water at 65°, formerly performed by continuous flow from a can 2 feet above the subject's head, is now commonly done by injection of 5 to 10 c.c. repeatedly from a syringe. Care must be taken not to use undue force. With the head 30 degrees forward, rotatory nystagmus toward the opposite side ordinarily occurs in from 30 to 60 seconds. This should be relatively equal for the two sides. The caloric test must not be done when the tympanic membrane is perforated. If the head is moved back 120 degrees, rotatory nystagmus resulting from stimulation of the vertical canal at 30 degrees changes to horizontal nystagmus in the same direction.

If no reaction occurs within four or five minutes, the labyrinth on that side is dead.

Good hearing is essential for the aviator, since he depends upon spoken or signaled orders by radio. Criticism of the high sustained tone of the radio beam signal, alleging that undue fatigue and occasional misinterpretation or even loss of the signal may occur, is not borne out by airline pilots or flight surgeons. Their recent experience fails to justify such criticisms, which seem based on observations made some years ago, when airplane noise was not screened and radio loudness was not subject to rheostat control. Effects of flight upon hearing depend on noise and vibration, and on changes in air pressure while ascending or descending. Noise and vibration from engine, propellers and exhaust are somewhat masked by the pilot's helmet and rubber-cupped radio head set. They are more severely felt by other flying personnel and passengers not so equipped. Cotton or rubber ear plugs may be used by these people. Sound-proofing of cabin type planes, now standard in all commercial transports, eliminates much of the noise damage caused by exposure in open cockpit models. Audiometer tests have shown gradual decrease in acuity for the higher frequencies in older pilots, but not significantly greater than in civilians of similar age groups. Exposure to these noises, which lie between 200 and 500 double vibrations, produces acute fatigue, and a sudden drop or "dent" around 4096 double vibrations. This has been ascribed to the vulnerability of the basal turn of the cochlea, directly exposed to the force of vibration through the middle ear, ossicles and round window. Repeated exposure to such fatigue has produced permanent losses around 4096 double vibrations in numerous well-authenticated cases.

Changes in air pressure interfere with hearing for the lower range of conversational tones. In ascent, pressure is greater inside the ear than outside; slight fullness is felt, and at about 500 feet (760 mm. Hg) the pressure is equalized and a clicking noise is perceived as the tube opens, letting out retained air. This is facilitated by swallowing or yawning. Above 500 feet elevation, the tubes open at approximately 425 foot intervals, up to 35,000 feet, when the air pressure is only 190 mm. Hg. During descent, if a speed greater than 300 feet per minute is reached, swallowing will open the tubes with increasing difficulty; increased air pressure in the nasopharynx tends to hold the tubes tightly closed. This occurs with a differential pressure of 80 to 90 mm. Hg, and causes severe pain, local congestion, especially of the drum membrane; transuda-

tion or hemorrhage into the middle ear; and low tone deafness of varying degrees, the condition known as acute aero-otitis media.

Repeated exposure to this hazard will lead to chronic thickening of the tympanic and eustachian linings, with eventual formation of connective tissue and considerable reduction of hearing for low tones. Catheterization or Politzerization of such ears gives rapid relief, but precautions to avoid introduction of nasopharyngeal bacteria must always be taken, to avoid infection of the sterile transudate in the tympanic cavity. Pilots—and passengers—should not be permitted to fly when suffering from acute rhinopharyngeal infections—sinusitis, tonsillitis and the like. Careful consideration of the anatomy and physiology of the eustachian tube, especially of its middle portion, has convinced flight surgeons that this structure, because of its valvular action during rapid descent, is far more important during flight than the middle ear. Tuttle has contrived, for passenger use, a small rubber balloon with wooden nasal tip, which is blown up by one nostril to produce sufficient pharyngeal pressure on swallowing to open refractory tubes. Certain foreign experimenters have attempted to secure accurate readings on the function of the eustachian tube by more or less elaborate manometric devices in the external auditory meatus, while others use magnifying otoscopes to measure the movements of the malleus and tympanic reflex during swallowing or the Valsalva procedure. Such experiment, forgetful of Toynbee, Lucae and Politzer, has seemed unnecessary to American flight surgeons. They suggest that lymphoid tissue about the middle portion of the tube is often an unrecognized factor in facilitating tubal obstruction, and attach little importance to intratympanic changes, unless serous aero-otitis media recurs frequently in the same individual.

Rapid negative acceleration (diminished gravity) as in power diving from a considerable height, drives blood toward the head, produces passive hyperemia of the eustachian tube and tympanic structures, increases intracranial pressure, and may conceivably produce minute hemorrhages in the end organs of hearing and equilibrium. These effects do not correspond with those of chronic fatigue, brought on by many hours of exposure to noise in open planes. While hemorrhages and eventual scarring may occur in fatigue cases, final results resemble ordinary conduction deafness except for the eventual drop at 4096 d.v. The picture is similar to that of occupational (traumatic) deafness from industrial noise exposure or work in compressed air locks. Ischemia of tubal and tympanic structures, produced by rapid climbing (increased gravity

or positive acceleration) is followed by a period of active hyperemia, leading eventually to the same effects as those more acutely caused by power diving (diminished gravity).

Intratympanic pressure changes play a distinct role in the onset of fainting due to rapid ascent to critical altitudes, adding their effects to those of anoxia above elevations of 15,000 to 20,000 feet. Use of the oxygen mask, and installation of sealed, low-pressure cabins in larger planes, will control such symptoms. The sudden shock of intratympanic deflation may well contribute the decisive factor to a dangerous loss of consciousness in high flight, especially in fighting maneuvers.

Airsickness, with characteristic pallor and sweating followed by nausea, vertigo and vomiting, may be due to excessive labyrinthine stimulation in rapid turning, banking or sudden descent; but visual impressions and visceral disturbances usually contribute to the clinical picture. As with sailors, familiarity with such vestibular, visual and somatic stimuli through training rapidly reduces sensitivity in all save a small group, who will ordinarily exhibit other signs indicative of emotional instability. Involuntary fear often accompanies vertigo, which is actually a temporary disorientation depending upon faulty interpretation of one or more of the stimuli received from the various components of the sense of equilibrium.

Gravity and motion in space are perceived through muscular, articular, visceral and cutaneous sensations, as well as through the end organs of vision and of the vestibular system. Faulty interpretations of somatic and labyrinthine impressions must be trained out of the pilot. This is especially important in instrument (so-called "blind") flying, where vestibular impressions may totally contradict the evidence of the instrument board. Pilots who have relatively insensitive labyrinthine reactions and are otherwise somewhat phlegmatic are less likely to experience airsickness, and will have less annoyance during rapid combat evolutions. Occasional civilian passengers may possess similar immunity. The vestibular apparatus is adapted to the perception of relatively rapid changes in equilibrium, particularly when the body is in motion. To the flyer, his vision and static or viscerosomatic perceptions of equilibrium (the "feel of the plane") are much more important.

Persistence in flight through turbulent air or into a head wind may, not infrequently, force the pilot into a wing-down position, due to faulty interpretation of his vestibular stimuli; he feels that he is sitting upright, but is actually leaning sidewise. This is cor-

rected by attention to the instruments. Similarly, on recovery from a spin the sudden sense of reversal of motion (as with the turning chair) may send the pilot into a second spin in an effort to recover his normal balance, a most dangerous error unless, again, he has learned to place entire confidence in his instruments and to disregard his own sensations. At high speed there is little or no time to allow faulty labyrinthine reactions to subside; instant reliance upon vision and instruments must prevail. Excessive vestibular sensations are always distinctly hazardous.

Toxic action of tobacco on the auditory nerve has long been recognized. Many pilots smoke excessively; nevertheless, aside from its acceleration of the pulse, moderate smoking off duty is probably harmful only to susceptible individuals. Smoking during flight by passengers is less agreeable at high altitudes, but is deleterious only to those with diminished tobacco tolerance, rapid heart or high blood pressure.

Alcohol, even in small concentrations, releases inhibitions and may lead to recklessness. It must not be consumed before maneuvers, particularly when high flight is involved, since alcohol itself produces tissue anoxia and thus appreciably lowers the aviator's "ceiling". Increased blood pressure and cardiac rate from alcohol add to the danger of hemorrhagic aero-otitis, especially in rapid descent.

Acute upper respiratory disease, because of active hyperemia in the nose and throat, creates a flight hazard which might add infection to ordinary aero-otitis. Personnel with severe colds, bronchitis, sinusitis or tonsillitis should not be permitted to fly. The reclining posture, as on sleeper planes, increases the danger of tubal closure, and passengers with sensitive ears or with colds are awakened during descent to night landings.

Temporary psychic instability—worry, family or financial trouble—predisposes to circulatory upsets and thus to interference with tympanic and labyrinthine function. A feeling of exhiliration along with motor weakness supervenes at such altitudes as 15,000 feet, and denotes incipient anoxia, which may call for the oxygen tube. The onset of such symptoms, similar to those of mountain sickness, is a prelude to actual vertigo and nausea, and is quickly relieved by rest and oxygen.

Indiscriminate use of ephedrine, and particularly of amphetamine (benzedrine) to secure shrinkage of the turbinates and tubal orifices is undesirable because of subsequent secondary engorgement

of these structures. Abuse of amphetamine also carries the danger of excessive psychic and circulatory stimulation. It is usually available for air passengers whose nasopharyngeal congestion is excessive, but must be employed cautiously.

Obviously, individuals suffering from high blood pressure or those with impaired coronary circulation have no business traveling by plane. Many do; but they undergo distinct hazards, and their fragile capillaries readily establish painful exudations, in spite of the slow ascent and descent prescribed for commercial airliners.

Gas embolism due to nitrogen bubbles released in the blood at high altitudes has not been considered a factor in the "blacking out" of consciousness which affects substratosphere fliers; this phenomenon is definitely ascribed to oxygen starvation. Hence there is no present reason to fear such embolic accidents as affecting the cochlea or the vestibular apparatus; nor is the civilian flying below 15,000 feet liable to any such decompressive hazards.

Much more detail might be included in the ear examination of pilots; but otologists must remember that the psychic, cardiac and visual examinations are even more important, and they take a lot of time. It would seem that under the pressure of training schedules flight surgeons of the army and navy have been doing an excellent job, and that their standards are relatively simple and workable. Certainly the fine physical condition of the majority of civilian air line pilots demonstrates that such standards of examination and rechecking have been effective over a good many years. Fatigue losses of hearing, due in general to noise exposure in older types of planes lacking sound-proofing precautions, has been the only severe occupational hazard suffered by this group. Younger pilots do not now seem to lose hearing so rapidly as did the now diminishing group of World War pioneer flyers.

Otologists generally should acknowledge their debt to America's pioneer flight surgeons—notably to Bauer, Tuttle, Armstrong, Campbell and Hargreaves—for practical clarification of many of the problems of aviation, for passenger and pilot alike. I should like to add a word of personal appreciation to Colonel Tuttle and Major Campbell for their assistance and suggestions in the preparation of this review.

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XXXII

DEVELOPMENT OF THE OTIC CAPSULE*

VI. HISTOLOGICAL CHANGES AND VARIATIONS IN THE GROWING BONY CAPSULE OF THE VESTIBULE AND COCHLEA

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The internal ear, a highly complex special sense organ, is housed in the bony otic capsule. The structure of this otic capsule is of interest first, from a purely scientific point of view, because its development and the structure of the adult bone differ in several respects from that of other bones of the body; and second, from a clinical and pathological aspect, since in certain types of functional impairment of the ear there occur as a rule certain types of pathology in the capsular bone. Such capsular pathologies as otosclerotic foci, ankylosis of the stapes, and capsular fractures have long been associated with impairment of auditory function. Much has been written, especially in later years, on the development and structure of the otic capsule. The localization of ossification centers has been described by a number of authors since the first account by Kerckringius[1] in 1670. The laterature on this subject is cited in the account by Bast[2] in 1930. The chronological order of events in the ossification of the capsule in the canal region was described by Bast[3] in 1932. Other structures such as the "fissula ante fenestram", "fossula post fenestram", pneumatic spaces and residual cartilages in the bony capsule have been described and their relationship to pathological processes have been discussed by various authors. The related literature is cited by Anson and Wilson,[4, 5] Anson and Martin,[6] Wilson[7] (1935), and Bast.[8-12] The literature on the pathology of the capsule such as otosclerotic foci and stape ankylosis is well summarized in the two volumes on otosclerosis presented in 1939 by the American Otological Society.[13] In 1939 Grove[14] summarized the literature on fractures of the otic capsule and petrous bone. Regarding the development and structure of the bone in the various parts of the cochlear and fissular portions of the bony otic capsule little information is available.

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*From the Department of Anatomy, the University of Wisconsin.

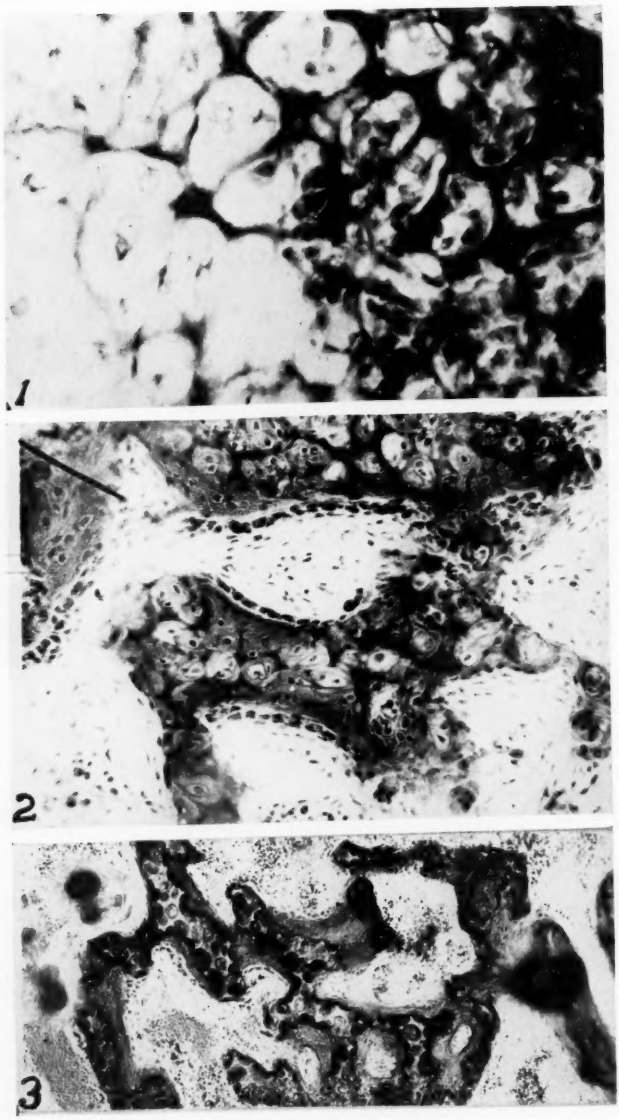
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Fig. 1.—Photomicrograph of portion of otic capsule of an 18-week-old human embryo (E. 38-147 mm. C.R.) showing enlarged cartilage lacunae with their necrotic cartilage cells at the left. At the right the cartilage lacuna are being invaded by osteoblasts.

Fig. 2.——Photomicrograph of portion of otic capsule of a 21-week-old human fetus (E. 21-183 mm. C.R.) in which the middle layer consists of irregular masses of the original but changed cartilage and excavated areas containing osteogenic tissue. The changed cartilage matrix has become calcified. The old lacunae are each filled with a variable number of osteoblasts each of which has already deposited true bone around itself. This combination of calcified cartilage matrix containing bone within its original lacunae has been termed intrachondrial bone.

Fig. 3.—Photomicrograph of the otic capsule of a 26-week-old human fetus (E. 2-23 mm. C.R.). This is a later stage than that shown in Fig. 2. Some endochondral or replacement bone has been deposited on the surface of the intrachondrial bone.

The purpose of this paper is to present chronologically the changes which occur during the process of development and to describe the structural make-up of the bony capsule in the region of the cochlea and fissula. It is hoped that such information may throw some light on the problems of petrous bone pathology.

The observations reported herewith are based on serial sections of the petrous bones of 61 human fetuses ranging in age from 18 weeks to term and of 18 infants ranging in age from 3 days to 6 years. In most cases both right and left ears were used. Thus approximately 50,000 sections were studied. These sections were projected at a magnification of about 50 diameters and the general characteristics noted. From this general analysis a representative series of 37 petrous bones was selected. To aid in the analysis photomicrographs were taken of three or four representative sections through each of these 37 bones. These pictures were mounted on cardboard and could thus be easily spread out on a table for making a comparative study. A sectional model was made of the petrous bone of a 30-week-old fetus at a magnification of 20 diameters. This model can be separated at eight different levels. At each level an accurate drawing shows the detailed structure and size of the several types of bone which make up the otic capsule.

The otic capsule is composed of three layers of bone: (a) the periosteal layer, (b) the middle or so-called endochondral layer and (c) the endosteal layer. The basic or fundamental layer is the middle or endochondral layer, and in it are the developmental and structural factors which differentiate the bone of the otic capsule from other bones of the body. The periosteal and endosteal layers develop in a manner much the same as in other bones.

Developmentally the early otic capsule is a cartilaginous one which at first encloses the early endolymph portion of the ear. The early cartilaginous capsule is bulky and devoid of its definitive form. Its definitive form is attained by several important changes. At about the 35 or 40 mm. stage the cartilage immediately surrounding the growing otic vessicle undergoes a dedifferentiation into a loose mesenchymal tissue which by the enlargement of its meshes becomes the periotic space. The formation of the periotic labyrinth is well described by Streeter.[15] The rest of the cartilaginous capsule enlarges and expands keeping pace with the expanding cochlea and semicircular canals. In the canal region some of the cartilage is removed by the vascular buds from the subarcuate fossa. This process was previously described by Bast[3] and will not be discussed here. The cartilaginous capsule reaches its definitive form between

the sixteenth and twentieth weeks of fetal life or between the 126 mm. and 18 mm. stages at which time the cochlea and canals reach their maximum size and at which time ossification of the capsule occurs. As previously described by Bast[2] the earliest ossification centers appear in the sixteenth week of fetal life and the last at about 20 or 21 weeks. In the cochlear capsule the ossification occurs between the sixteenth and twentieth weeks or between the 135 mm. and 170 mm. stages.

It is the ossifying cartilaginous otic capsule that gives rise to the middle or so-called endochondral layer of the finished otic capsule. In the cochlear region the form and size of the middle layer are the same as those of the definitive cartilaginous capsule from which it arises except at the medial side of the cochlea where the cartilage capsule continues to grow to form the anlage of the future petrous apex. The outer or periosteal layer is formed, from the cambium of the periosteum and the inner or endosteal layer from the cambium of the endosteum, in a manner much the same as for long bones.

The details of the osteogenesis of the middle layer of the otic capsule has been described (Bast[2]) and only the essential steps of the process in the cochlear portion of the capsule will be described here.

The earliest ossification center appears at about 16 weeks in the capsule over the base of the cochlea near the round window. During the next week or two, five ossification centers appear around the internal auditory meatus and one in roof of the cochlea. From these centers ossification progresses very rapidly so that by the twenty-first week the capsule around the cochlea is well ossified except for a small area (the fissular area) between the vestibule and the cochlea and an area medial to the cochlea where the cartilage continues to grow to form the petrous apex.

In the process of ossification the following changes occur:

1. The cartilage cells enlarge and the matrix between the cells becomes calcified. Most of the cells enlarge so much that the wall or matrix between the lacunae breaks down at spots and the lacunae communicate with each other. The cartilage cells then shrink and disintegrate.

2. With the calcification of the matrix the perichondrium becomes the periosteum. The cells of this periosteum which lie next to the calcified cartilage matrix change into osteoblasts. Some of

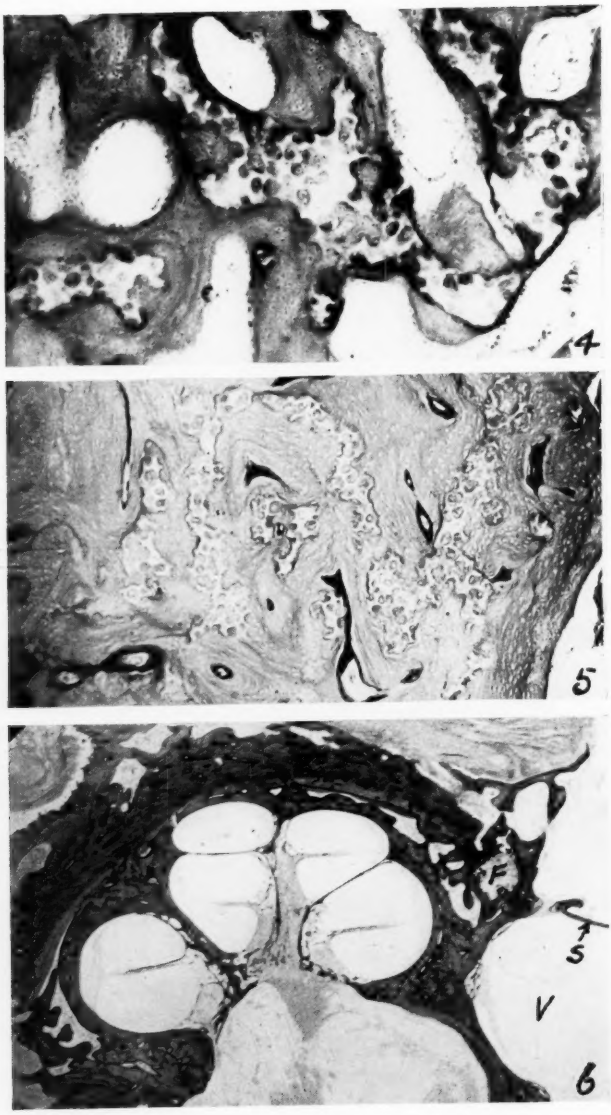
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Fig. 4.—Photomicrograph of portion of the middle layer of the otic capsule from a term fetus (B-143). Considerable replacement (endochondral) bone is seen around the intrachondrial bone but the bone marrow spaces are still prominent.

Fig. 5.—Photomicrograph of the middle layer of the otic capsule of an adult. Note the increase of the replacement bone (endochondral) and absence of bone marrow.

Fig. 6.—Low-power photograph of the cochlear portion of the otic capsule of a 7½-month-old child (B-120-R). The middle layer is already compact and similar to that in the adult. There is, however, considerable resorption of bone between the middle and periosteal layer. Subsequent ossification within these resorbed areas knits the two layers of bone firmly together. *S.* Stapes. *V.* Vestibule. *F.* Fissula ante fenestram. Note that the bone around the fissula belongs to the middle layer.

these osteoblasts begin to form periosteal bone and others wander into the opened and enlarged cartilage lacunae. Anywhere from one to eight such osteoblasts will enter one lacuna where they will deposit bone around themselves so as completely to fill the lacunae with bone. While this goes on osteogenic buds will also invade the changed cartilage, opening large spaces but leaving considerable masses and strands of the calcified cartilage and the enclosed lacuna containing the newly acquired bone. At the right in Fig. 1, the osteoblasts can be seen filling the lacunae, while at the left the enlarged cartilage lacunae still contain necrotic cartilage cells. In Fig. 2 vascular buds have removed much of the changed cartilage but large islands of the calcified cartilage can be seen with their lacunae filled with many bone cells surrounded by bone. These masses or strands are the remnant of the original cartilage matrix which is now calcified and whose lacunae are filled with true bone. In previous accounts by Bast and Anson this type of tissue has been referred to as intrachondrial bone (cartilage islands or "globuli ossei" of other authors). It consists of the calcified cartilage matrix with true bone within its lacunae. This intrachondrial bone formed in the first week of the ossifying process is a permanent tissue and remains throughout life as a part of the middle layer of the otic capsule (Fig. 5).

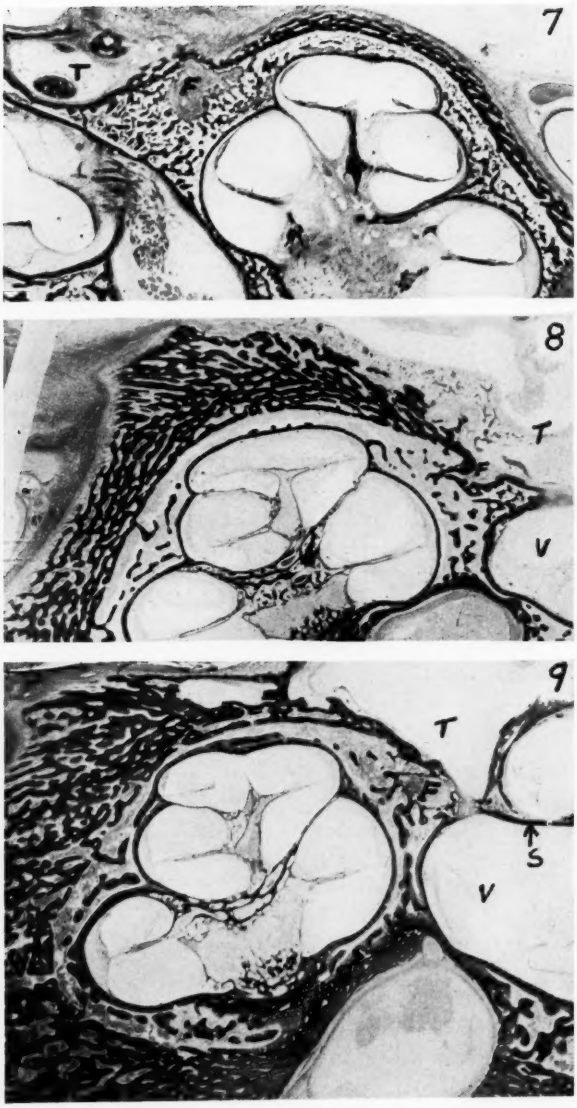
3. Little change occurs in the middle layer from the twenty-first to about the twenty-sixth week. Osteoblasts line up at the margin of the excavated spaces on the surface of the intrachondrial bone (Fig. 2). These osteoblasts then deposit endochondrial or replacement bone on the surface of the intrachondrial bone. The early steps in this process are shown in Fig. 3. Endochondrial ossification is slow during the rest of the fetal life and at birth the middle layer may still have large marrow spaces (Fig. 4). At about birth the process speeds up and within a few months the ossification of the middle layer is pretty well completed (Fig. 6). The composition of the middle layer attained at this stage is retained throughout life (Fig. 5). Up to about the seventh month of postnatal life, the boundary of the middle layer is quite distinct from the periosteal layer and as a result it is not firmly united to the rest of the petrous bone. To accomplish this union resorption of part of the middle and periosteal layers takes place and these excavated areas are then filled with new bone, thus knitting the two layers firmly together. In Figs. 10 and 11 the middle layer is clearly delimited from the other layers. In a 7-month-old child (Fig. 6) there is marked erosion between the middle and periosteal layers and the boundary is less distinct. In Fig. 12, taken from a 3-year-old child, the new

bone has filled such excavated areas and the line of demarcation between the middle and periosteal bone is largely obliterated, the narrow spaces are reduced to a minimum and all three layers are closely fused into a compact mass of bone which normally persists throughout life.

In the evaluation of the significance of the endosteal, periosteal and middle layers of the otic capsule certain observations on the time and speed of their development and on the role they play in the composition of the capsule seem worthy of consideration.

1. The endosteal and periosteal layers begin to form from the internal and external cambium as soon as the matrix of the cartilage of the middle layer becomes calcified. That is, they begin to develop early in the ossifying process. The endosteal layer increases in size but very little after it is first laid down (Figs. 7-10). Throughout life it remains as a thin layer which is fairly uniform in thickness in all parts of the capsule.

The periosteal layer begins at the same time but develops very rapidly into a very prominent part of the otic capsule. A glance at Figs. 7 to 9 will show that it develops rapidly between the onset of ossification up to the twenty-seventh week of intrauterine life. Between the twenty-seventh week and fortieth week or term there is little increase in size. (Compare Fig. 9 with Figs. 10 and 11). Thus the periosteal bone reaches its maximum growth at about the time when the endochondral bone of the middle layer begins to be formed. During the period between the twenty-seventh week and term the periosteal bone does not expand but there is some resorption and rebuilding so that at birth (Fig. 11) it is somewhat denser than at the twenty-seventh week. It appears that at about the twenty-seventh week there is a shift of bone deposition from the periosteal layer to the formation of the endochondral or replacement bone around the intrachondrial of the middle layer. Even this endochondral bone formation is slow so that by the thirty-ninth week (Fig. 10) the middle layer still has much bone marrow and the bone spicules are only slightly larger than at 27 weeks (Fig. 9). During the last week of fetal life the bone marrow is more rapidly replaced by endochondral bone, as can be seen in Fig. 11. Immediately after birth the marrow of both the middle layer and periosteal layer is rapidly replaced by bone. Thus in a 7-month-old child (Fig. 6) both layers are made up of compact bone except for re-excavated areas between the two layers. By the third year (Fig. 12) all the layers consist of compact bone and the lines marking the three layers are obliterated. A bird's-eye view of these changes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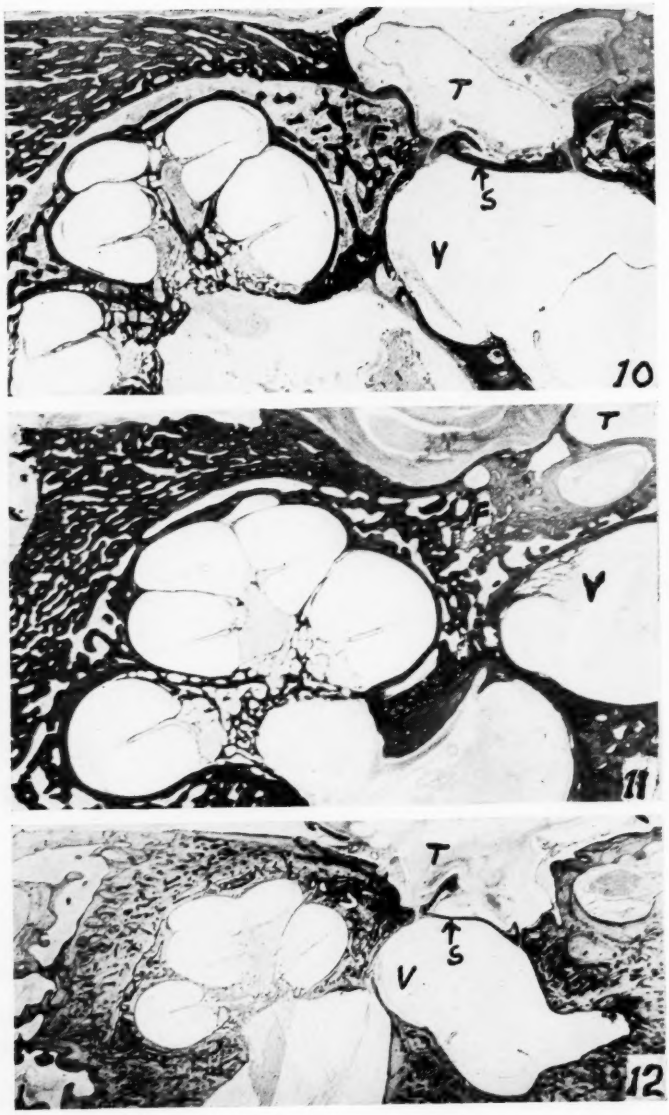
Figs. 7, 8 and 9.—Photomicrographs of the cochlear portion of the otic capsule to show the degree of development of the endosteal, periosteal and middle layer of bone. Note that in the region of the fissula the capsule is made up almost entirely of the middle layer of bone. *F.* Fissula ante fenestram. *V.* Vestibule. S. Stapes. *T.* Middle ear.

Fig. 7.—Fetus 21 weeks old (E. 21-L-183 mm. C.R.). The middle layer consists of spicules of intrachondrial bone and primitive bone marrow spaces. The periosteal bone is developing rapidly but is still thin.

Fig. 8.—Fetus 24 weeks old (E. 51-210 mm. C.R.). The middle is essentially the same as in Fig. 7. The periosteal layer has reached considerable proportions.

Fig. 9.—Fetus 27 weeks old (E. 89R-243 mm. C.R.). At this stage the endochondral (replacement) bone is beginning to be deposited around the spicules of intrachondrial bone. The periosteal bone has about reached its adult proportion and some resorption is taking place in it on the middle ear side.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Figs. 10, 11 and 12.—Photomicrographs of the cochlear portion of the otic capsule to show the degree of development of the endosteal, periosteal and middle layers of bone. Note that in the region of the fissula the otic capsule is made up almost entirely of the middle layer of bone *F.* Fissula ante fenestram. *S.* Stapes. *V.* Vestibule. *T.* Middle ear.

Fig. 10.—Fetus 39 weeks old (B-124 R.). Very little endochondral (replacement) bone has been deposited around the intrachondrial bone spicules. The bone marrow spaces are still large in this middle layer. The periosteal layer is much the same as at 27 weeks.

Fig. 11.—Term baby (B-122 R.). The marrow spaces of the middle layer are rapidly reduced in size due to the rapid deposit of endochondral bone on the spicules of intrachondrial bone. The periosteal layer is more compact.

Fig. 12.—Three-year-old baby (B-96). All layers of bone are now compact and the line of demarkation between them is indistinct.

can be had by comparing Figs. 9 to 12. The marked paucity of bone in the middle layer up to the end of intrauterine life is very striking.

2. Another observation that should be stressed is the relative thickness of the three layers of bone in the otic capsule. The endosteal layer is always thin and fairly uniform. On the other hand there is a variation in the thickness of the middle and periosteal layers. Most of these variations seem of little significance and can be explained on the basis of irregularities of the periotic labyrinth which the capsule surrounds or the irregularities of the surface of the petrous bone.

Special attention should be called to the area anterior and medial to the oval window and between the vestibule and cochlea. This area is often referred to as the fissular area for it is here that the fissula ante fenestram occurs. The area of the fissula ante fenestram is shown at *F* in Figs. 7 to 11. Here the capsule is made up almost entirely by the middle layer and only a very thin periosteal layer separates the middle layer from the tympanic cavity. It is further of interest that it is in this region that otosclerotic foci are usually found and that ankylosis of the stapes begins. In 1940 Bast[12] showed that residual cartilages, neoplastic cartilages or defective ossifications occurred at various specific spots in the otic capsule. The unstable spots always occur in the middle layer and where it is prominent. The indication, therefore, is that this middle layer is the more unstable type of bone and it may be subject to subsequent changes. Otosclerotic foci have been associated with such areas and it would be of interest to know if any of the other pathological processes may have their starting points in these areas.

SUMMARY

The otic capsule consists of three layers of bone, the periosteal, the endosteal and the middle layer. The histogenesis of each of these layers is described.

The relation of the middle layer to pathological processes is discussed especially in the region of the fissula ante fenestram where the capsular wall is made up almost entirely of the middle layer.

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XXXIII

NUTRITIONAL DEFICIENCIES IN OTOLARYNGOLOGY*

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KANSAS CITY, MO.

For years I have been convinced there was some nutritional or biochemical background for many otolaryngological complaints. So many symptoms complained of by the patient could not be explained on the basis of infection, deformities, or new growths. Therefore, I suggest that the following symptom complex be considered a syndrome: (1) postnasal discharge without sinus infection; (2) sensation of stuffiness or obstructed nose without obstruction; (3) sinus pain without sinus infection; and (4) fatigue without organic findings.

These and many other symptoms, such as auditory neuritis without discoverable etiology and nervous symptoms psychoneurotic in type without positive findings, are frequently present.

Postnasal discharge is the most common; it causes much clearing of the throat, cough, sensation of fullness and sometimes actual choking sensations. Direct examination usually shows only a mucoid secretion in the postnasal space. Anterior rhinoscopy shows mucous bridges from the septum to the lateral wall. Findings are negative for sinus infection and there is no real obstruction from deformity or new growths.

Nasal fullness or obstruction is frequently alternating from side to side and is worse when lying down and in the early morning. Again, direct examination is not very illuminating. Frequently although there is a septal deviation to one side causing the obstruction, the patient complains of his worst difficulty on the opposite side where there is an actual concavity.

The findings are negative or only show some of the above where sinus pain occurs without sinus infection. In these cases one always looks for nerve pressure or vacuum pain. The usual run of diagnostic measures such as transillumination, X-ray, and sinus washings are negative. The pain may be worse on one side and the obstruc-

*Presented before the Middle Section of the American Laryngological, Rhinological and Otological Society, Jan 21, 1942, St. Louis, Mo.

tion worse on the other. There is often fatigue to the point of complete exhaustion. The symptoms are without ample corresponding physical findings. The same is true of auditory neuritis. There are often no obvious foci of infection, no vascular or blood changes yet the patient is losing hearing.

More than twenty years ago I remember hearing the late J. A. Stucky, of Lexington, Ky., plead before the national societies to make nutritional investigations in otolaryngological troubles before resorting to surgery which might be of doubtful value.

Isaac Jones[1] has recently published a masterful dissertation on vitamins worth the serious reading of all those in our specialty.

The basis of my report is more than 3,000 diet histories of office patients in the last six years.

The reply to the question, "Do you eat a well-balanced diet?" is nearly always "Yes." One woman, the wife of a civil engineer and above the average in intelligence, admitted eating a half-pound of candy daily in addition to large quantities of white sugar in other foods and also drinking twenty "cokes" per week. She stated she was not conscious of any overindulgence in carbohydrates and that sweets gave her a lift. Her chief complaints were headache, postnasal drip, nasal obstruction, and fatigue—the syndrome suggested. Of course, she was tired all of the time often to the point of complete exhaustion. She had other complaints too numerous to report. She took a mineral oil cathartic daily. She had consulted her family physician because of nervous exhaustion. He did not take a diet history, but relied upon an intelligent patient's statements that her diet was excellent and well-balanced. He gave her a sedative and advised rest. When she did not improve she diagnosed her own case as "sinus disease" and consulted me. Needless to say she had no actual sinus suppuration, but what she lacked in sinus disease was made up in nasal symptoms. This was sufficient excuse to justify keeping her under observation for a few months until I was sure her nutrition was improved and some of her bad habits corrected.

It has now been several years since she first consulted me and to this day she is convinced all of her body ills were due to "sinus disease". She has since remained well from all nasal and other symptoms. Her case is typical of hundreds I have seen with this syndrome.

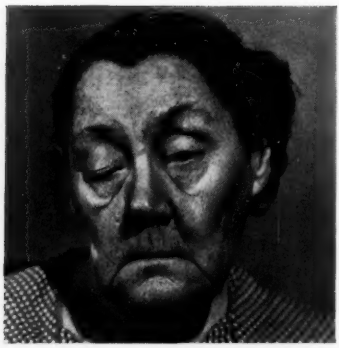

Fig. 1.—Subclinical pellagra. Paralysis of all ocular muscles. Complete ptosis right eyelid; partial ptosis of left. Bilateral auditory neuritis.

Fig. 2.—Ichthyosis-like skin in case of subclinical pellagra.

It was most difficult in this woman's case, as it has been in many others, to get them to discontinue taking mineral oil. I explained that mineral oil renders practically all vitamin A in the intestinal tract insoluble, hence unusuable. I made it plain that this vitamin is essential to the mucous membranes of the nose and sinuses. As stated by Curtis and Horton,[2] amounts of mineral oil as small as 15 to 30 c.c. are able to remove the carotene (pro-vitamin A) from the normal diet if the oil comes in contact with the food substances containing the carotene.

It is my opinion that mineral oil not only renders vitamin A insoluble but also interferes with the absorption and utilization of many or all of the other vitamins.

Morgan[3] says: "It would not be too unfair to say that in some respects mineral oil has earned its niche in the section of toxicology rather than in pharmacology. The wide use of liquid petrolatum seems to be based on empirical considerations which make little sense when examined critically. Its chemistry is to say the least uncertain and its pharmacologic action a matter of dispute.

"Fifteen years ago Burrows and Far did some experiments which demonstrated that mineral oil was fatal in every case when it was fed to rats. Perhaps, this phenomena is related to what Manwaring calls 'vitamin blockade'."

What applies to mineral oil may also apply to many other cathartics and physicians should never encourage this pernicious habit. We should think as sharply before prescribing a regular or P.R.N. cathartic as we do before giving a narcotic prescription.

The two chief dietary faults are excessive use of white sugar and white flour. The combination of white bread, sweet desserts, sweet fountain drinks, pastries, and the like forms the etiological background for many otolaryngological complaints. Many other general symptoms and psychoneurotic conditions follow in the wake.

The chief foods absent from most diets are green, leafy, raw vegetables and fresh fruits. The diet histories seem to show that most patients have ample meat. It is a well-known fact that Stefansson, the Arctic explorer, lived seven years on an exclusive meat diet and was in perfect health. I feel that it is more the overindulgence in certain foods that is chiefly to blame. We are a sweet-eating nation, each person consuming 115 pounds of sugar annually.

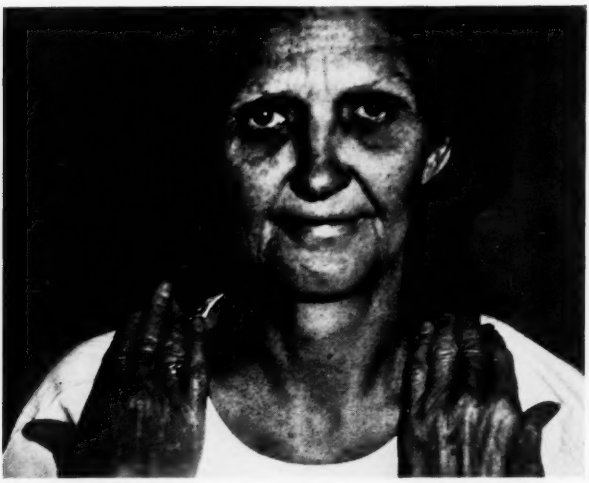

Fig. 3.—Patient with subclinical pellagra, showing marked pigmentation of skin. Patient died twenty-four hours after picture was taken.

What may be an adequate diet for one individual may not be sufficient for another. The patient must be studied as an individual. Different racial strains require different foods. We, in this country, are frequently a mixture of all of the European racial strains, but there is usually a dominant strain.

Dental caries is almost unknown among the Eskimos or for that matter in any of the untouched natives. When refined foods (chiefly white flour and white sugar) were introduced into their diets, the percentage of dental caries rapidly increased. It requires several generations to lose this stored strength. It is a well-known fact that the Southern negro develops much less dental caries than the poor whites who consume practically the same foods. Medical teaching today and in the recent past generations considers micro-organisms the primary cause of disease. It is possible that the next generation will place bad nutrition and bad habits as the primary cause and micro-organisms as the secondary.

In the past when nutritional disturbances were the obvious cause of otolaryngological complaints, I referred patients back to their family doctor. I found this was not satisfactory for they were not interested in what they considered "minor complaints". So

often the report was either negative or that the patient was neurotic. To be sure, if the patient were diabetic, nephritic, or had heart disease, a scientific regime would be outlined with great care.

We must remember that we are physicians first and specialists later. We must not try to unlearn our clinical medicine.

One of my patients, a woman, an author and teacher, aged 35 years, had in addition to this syndrome severe neuritis in her right arm, fatigue to the point of complete exhaustion, chronic sore throat, and other symptoms.

She had consulted a number of excellent physicians in her community. Her blood, urine, heart, chest, and general physical examinations were negative. When she did not improve, after following directions, she went to a clinic in a nearby city.

Again, the tests were negative and the director of the clinic in summarizing her case told her: "There is absolutely nothing wrong with you. You are nervous, a neurotic and probably sexually unhappy." In describing this interview to me later she said: "It acted as an exploding bomb to finish shattering my already tense nervous system."

Even a superficial dietary inquiry would have revealed her trouble. Because of her intelligence and her desire to get well she cooperated in every way and in only a few weeks was entirely well.

Field, Parnell, and Robinson[4] state: "Pellagra is not confined to those two groups, alcoholic addicts and the Southern poor, whose dietary habits are notoriously bad and among whom the severe manifestations of the disease are especially common. Lesser deficiencies not accompanied by florid dermatitis, diarrhea, or dementia are common in the average Northern population. They are the cause of important impairment of health.

"Many have commented on the vitamin content of the modern American diet, due to an increasing consumption of sugar and refined flour, with an ensuing decreased consumption of vitamin-containing foods.

"Many of our patients have had access to, but have not selected, an adequate diet. People with poor appetites tend to select high carbohydrates food of low vitamin content.

"It is unfortunate that most of the descriptions of pellagra have emphasized the severe forms of the disease. Patients without

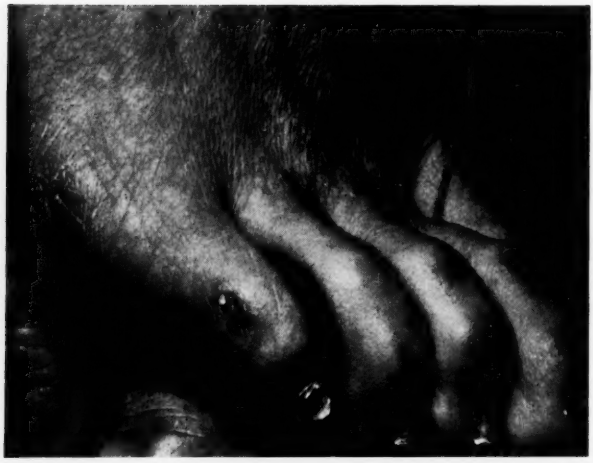

Fig. 4.—Hyperkeratosis over pressure joints in chronic pellagrous dermatitis.

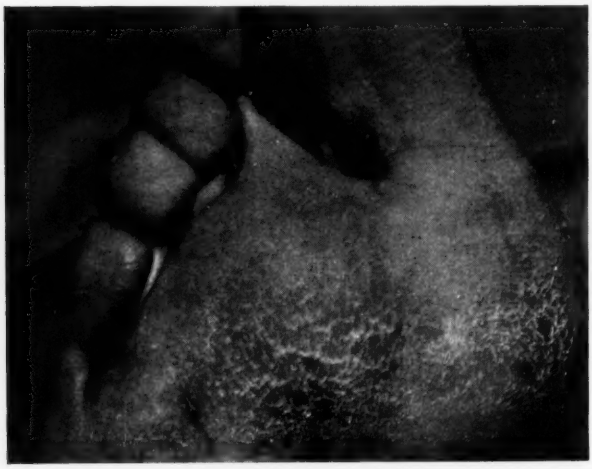

Fig. 5.—Hyperkeratosis over pressure joints in chronic pellagrous dermatitis.

the fully developed picture actually constitute a majority of pellagrins."

One of the characteristics of chronic pellagrous dermatitis is hyperkeratosis (Figs. 4 and 5). Common sites for hyperkeratoses are over skeletal pressure points such as the knee, elbow, instep and front and back of the ankle. A vitamin C deficiency appears in some cases and plays a role in the pigmentation. Another common skin manifestation is an ichthyosis-like change, which has received little attention (Figs. 2 and 6). Sometimes the skin is uniformly smooth and shiny. I have also observed that the mucosa of the nasopharynx often appears as though it had been shellacked.

Riboflavin deficiencies are frequently manifested by the mucosa of the lips showing superficial transverse fissures and scaly desquamation in the nasolabial folds of the vestibule of the nose and ears. There may also be seborrheic lesions scattered over the face; fissures at the angle of the mouth and over the main part of the lips, the latter showing in the form of vertical fissures.

In the last few months, I have been giving pantholin (Lilly) intravenously. Pantholin is pantothenic acid stabilized with calcium. While pantothenic acid cannot be detected in the blood a few hours after it has been given, it does increase the riboflavin as much as 20 to 30 per cent and, I believe, is indicated in all riboflavin deficiencies. I prefer to report later on final results, but they seem very satisfactory at this time.

Because the patient is in moderate or good economic status is no assurance that he eats the proper vitamin-containing protective foods. The name that is attached to this nutritional deficiency is of no great moment. I prefer the term "subclinical pellagra", which carries sufficient drama to make both the patient and the physician alert.

I want to emphasize four well-known factors that deplete the body of vitamin B Complex: (1) excessive alcohol, (2) excessive smoking, (3) excessive intake of carbohydrates, and (4) the pernicious habit of cathartics.

England has recently fortified margarine with vitamin A and flour with calcium and vitamin B. Our government has suggested to bakers that vitamin B, iron, and calcium be added to the highly processed white flour.

In addition to the above, recently we have all received from the National Council on Nutrition a recommendation that all salt

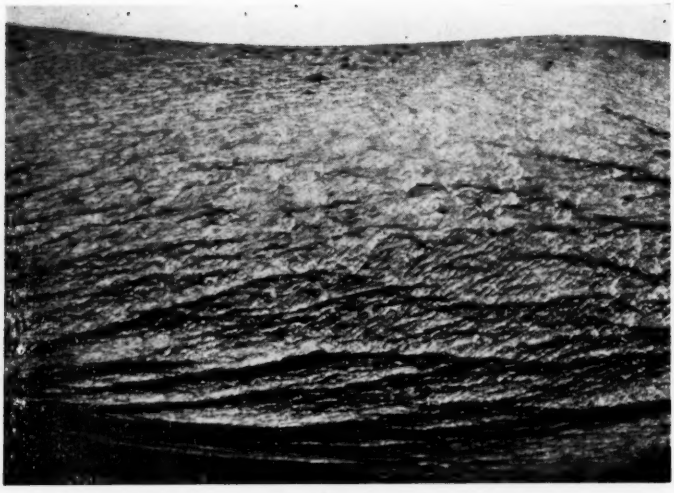

Fig. 6.— Ichthyosis-like skin in subclinical pellagra.

be iodinized. Persons who consume unusually large quantities of salt (sodium chloride) in the diet or those who live in cities supplied with chlorinated water may need an additional amount of iodine either in other foods or as a medication. This is due to the chemical law of halogen displacements. The displacement activity of any halogen is in inverse ration to its atomic weight. The following are the halogen weights:

Fluorine	19
Chlorine	35.4
Bromine	79.92
Iodine	126.92

All can displace iodine. As stated above this is especially true of chlorine as its weight is only about one-fourth that of iodine. It is on this principle that sodium chloride is given in larger doses in bromine poisoning with prompt recovery even though there is not such a great deal of difference in the atomic weight.

Iodine deficiency is frequently manifest by hypertrophied lymphatic nodules in the pharynx, nasopharynx, and cervical regions.

It is highly essential that vitamins be given by mouth until the diet can be regulated and in some of the more urgent cases intravenously.

I use multicebrin (Lilly) almost exclusively for oral administration because it contains the optimal daily requirement.

In cases of auditory neuritis I give nicotinicamide, 100 mg., intravenously, once a week in addition to the oral administration.

There is no doubt that the public generally is taking too many vitamins and not paying sufficient attention to their dietary habits. I insist that my patients take 5,000 to 10,000 units of vitamin A daily in the form of green and yellow vegetables. I insist upon fresh fruits three times daily; I do not as a rule restrict meats but I do suggest liver at least once a week and seafoods often. I try to have them discontinue all sweet desserts and substitute honey for sweetening on cereals and in coffee. Cereals should be only the whole or the fortified grain.

CONCLUSIONS

1. Many otolaryngological complaints listed under the syndrome such as postnasal discharge, obstruction to nasal breathing, headaches, and fatigue are practically always due to a nutritional disturbance.

2. There has been ample work done on auditory neuritis to show that nicotinic acid, and perhaps other factors of vitamin B, are important in its management.

3. These patients are very frequently diagnosed as psychoneurotics or at least are considered highly nervous when their nutrition is at fault.

4. To investigate the nutrition properly a careful diet history must be taken.

5. The chief faults leading to the deficiency are in overindulgence in the more refined foods and in the inadequate intake of green leafy vegetables, raw fruits, and proper meats.

6. There are no practical laboratory tests to determine accurately the vitamin deficiency. Clinical observation is our best diagnostic aid. There is usually a multiple vitamin deficiency, if vitamin deficiency exists at all.

7. One must individualize patients as to race, age, occupation, etc., in outlining an adequate diet.

8. All cathartics must be banned. The proper foods and a little perseverance eliminate all necessity for this pernicious habit.

In closing let me emphasize again that we must work out our own salvation with patients having otolaryngological complaints. The family physician should not be expected to work out our problems. We must be ever alert to see beyond the horizon of our speculi.

Since presenting the above report, we have given over 700 intravenous doses of the following clinical material.*

Riboflavin	2 mg.	per c.c.
Pyridoxine hydrochloride	1 mg.	
Pantothenic acid (as the calcium salt)	1 mg.	
Nicotinamide	100 mg.	

We have added to the above 50 mg. thiamine hydrochloride.

The indications for this line of treatment have been the syndrome discussed above and auditory neuritis, the results of which I shall report more in detail later. However, I am convinced that the results obtained by using the above combination are more satisfactory than when nicotinamide was used alone, intravenously, or when I depended upon oral administration.

I am of the opinion that where vitamin administration is indicated, in the more extreme cases, it is usually in a patient whose body tissues have been depleted of the stored vitamin and in whom the absorption from oral administration is entirely inadequate.

I also find that eight to ten intravenous doses, over a period of three to four weeks, are usually needed to replenish the depleted vitamins. Oral administration may be ample thereafter for maintenance.

I still have sufficient material to carry on these investigations for several months; however, due to unforeseen events, such an ampoule as I have mentioned will not per se be placed on the market for the present. I find that I can make up my own combinations from prodcts now on the market (nicotinamide, 100 mg. in 2 c.c. with pantholin [Lilly] 50 mg. per c.c.) but it is not as convenient as the "clinical" ampoule.

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*Supplied by the Eli Lilly and Company, Indianapolis.

My hope is that we shall eventually have not only a high potency vitamin B complex, but also a multiple vitamin ampoule for intravenous use.

1110 PROFESSIONAL BLDG.

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XXXIV

OTITIS EXTERNA*

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SAN ANTONIO, TEX.

Otitis externa constitutes one of the most frequent conditions encountered in otologic practice and for this reason, what might ordinarily be considered a most prosaic subject becomes of prime importance. So far as incidence is concerned, it has been variously estimated that otitis externa constitutes from 5 to 40 per cent of all cases encountered in otologic practice, the wide variation in figures being due to sectional differences in temperature and humidity. The term includes inflammatory conditions of the externa auditory canal as well as of the pinna. The following outline will be useful in the clinical classification of the various types of otitis externa:

I. Traumatic

II. Thermal
- A. Heat
  - 1. Dermatitis ab igne
- B. Cold
  - 1. Frost-bite
- C. Solar
  - 1. Dermatitis e solare

III. Due to radiant energy
- A. Ultraviolet
- B. X-ray
- C. Radium

IV. Chemical
- A. Simple dermatitis of chemical origin
- B. Dermatitis medicamentosa
- C. Dermatitis venenata (see also 4A and 6A)

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*Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Jan. 23, 1942, Atlanta, Ga.

V. Infections
  A. Pyogenic
  B. Nonpyogenic
  C. Mycotic
    1. Pyogenic
      a. Staphylococcus
        1) Impetigo contagiosa
        2) Pyogenic dermatitis
        3) Furunculus (abscess, acute)
      b. B. pyocyaneus
      c. B. coli
      d. B. friedländer
      e. Pneumococcus
    2. Nonpyogenic
      a. Streptococcus
        1) Cellutitis, acute
        2) Lymphangitis, acute capillary
        3) Erysipelas
    3. Mycotic (otomycosis)
      a. Due to budding forms (blastomycetes)
        1) Saccharomycetes
        2) Torulae
        3) Moniliae
        4) Coccidioides
        5) Blastomycetes
          (Cryptococcus dermatitidis gilchristi)
        6) Oidiae
      b. Due to filamentous forms
        a. Aspergilli
        b. Penicillia
        c. Mucors
      c. Due to higher bacterial forms
        a. Actinomycetes
        b. Nocardiae (acid-fast, non-acid-fast)

D. Tuberculous

E. Syphilitic

F. Leprous

VI. Allergic (causes numerous, external and internal)

VII. Dermatoses

A. Seborrheic dermatitis

B. Eczema

1. Exudative

2. Nonexudative

Traumatic otitis externa may be due to a number of mechanical causes. One of the most frequent is dressing the ear with gauze or using gauze to grasp the pinna and to retract it in mastoid operations. Gauze seems to be particularly well adapted to injuring the covering layer of epithelium of the ear unless great care is used when it is employed. Avoidance of harsh dressings or undue traumatism in cleaning the ear is recommended, for when epithelial denudation occurs, secondary infection easily becomes established, and healing is notoriously slow in such instances. The liberal use of petrolatum in postoperative mastoid dressings is a valuable prophylactic against such occurrences, as well as being of advantage in lessening the pain of such dressings.

The thermal types encountered are those due to heat and cold effects, a typical example being that type of inflammation which follows the effect of heat in the use of the actual cautery. The effect of sunlight on the skin of the ear is very largely a combination effect of heat and actinic rays acting coincidentally. Frost-bite is a common sight in winter in many localities. The effect produced by excessive cold on the tissues is precisely the same as that of heat, and varies with the intensity of the cold and the duration of its application. Hyperemia, vesiculation, bleb formation, and even necrosis of tissue constitute the sequence of pathologic changes in frost-bite and also in heat effects.

Radiant energy produces hyperemia, edema, exudation of serum, exfoliation of epithelium, or even destruction of tissue, depending on the intensity and duration of its action. The ultraviolet ray will readily produce a burn of the skin of the ear, and the X-ray, which has such a great field of usefulness about the ear, may produce

inflammations of an annoying and persistent nature. The reaction following the injudicious use of the X-ray about the ear is not immediate, but comes on after a period of from ten days to two weeks, and sometimes sooner. Radium reactions like those produced by the X-ray are somewhat delayed in making their appearance. They too are likely to be quite painful and persistent and at times quite destructive.

Simple chemical dermatitis may be produced by the effect of certain chemicals on the skin of the pinna or external auditory canal. There is only a narrow line of demarcation separating this type of otitis externa from dermatitis medicamentosa, dermatitis venenata, and allergic dermatitis. Often these types merge one into the other or overlap so that precise classification is not always possible. The term chemical dermatitis is reserved for that type of dermatitis which is due wholly to the effect of a chemical irritant with no implied degree of hypersensitiveness of the skin being present. A typical example would be the reaction following the application of trichloracetic acid or nitric acid to the skin. The local application of mercurials in persons taking iodine or iodides internally, or vice versa, results in the production of a violent reaction which is truly a chemical dermatitis.

Dermatitis medicamentosa approaches the allergic state very closely, but is different from it. It is a well-known fact that patients with heavily pigmented skins tolerate local applications of many drugs in higher concentration than persons with less pigmentation. Otitis externa may be induced and perpetuated indefinitely if this simple fact is not borne in mind. A drug may produce inflammation of the skin of the ear or external auditory canal and fail to do so in other parts of the body.

Dermatitis venenata is a time-honored term reserved for the designation of inflammatory reactions due to contact with poison oak or poison ivy. In this instance there is a degree of susceptibility present in the affected person. Therefore, this type of reaction is definitely in the group of allergic phenomena and is a typical example of a local reaction in a hypersensitized person.

Otitis externa due to infections is divisible into three classes, the nonpyogenic, pyogenic, and mycotic types. It is this group with which we are chiefly concerned as it supplies the greatest number of cases of otitis externa. Under the nonpyogenic classification are included cellulitis of the external ear due to bacterial causes. Erysipelas is properly included under this heading.

Pyogenic affections of the external ear provide a high percentage of all cases of otitis externa. The action of pyogenic bacteria on the skin of the external ear manifests itself in a number of ways; the furuncle, pyogenic dermatitis, and impetigo may be cited as illustrative of the pleomorphic character of the lesions produced by the staphylococcus.

The organisms concerned in the production of infectious types of otitis are the common pyogens such as the staphylococcus, colon bacillus, and Friedländer's bacillus. The Bacillus pyocyaneus is a rather frequent offender.

In the nonsuppurative types the chief reactions in the tissues are swelling and edema. Such conditions are usually designated as cellulitis of the external ear. Erysipelas comes in this category for it is nothing more than a diffuse capillary lymphangitis due to a specific type of streptococcus. Extension of pyogenic as well as nonpyogenic inflammation to deeper structures such as the cartilage of the ear can occur as a complication. This type of complication is most likely to occur following incision into the skin of the canal or pinna through an infected area.

Mycotic otitis externa is the type of external otitis most often seen in the South and Southwest. It is due to the invasion of the skin of the ear with molds, and while it is more frequent in moist, warm climates, no region is exempt for it can occur in any locality where conditions of temperature and humidity support the growth of molds. There is a seasonal variation in incidence as well as in the intensity of mold infections as cool weather inhibits the growth of molds, which, if sufficiently inhibited, cease altogether. There is no mystery about the organisms which cause otomycosis. They are divisible into three chief groups which are easily remembered: the budding, filamentous, and higher bacterial forms.

Infection with molds may be complicated by mixed infections with pyogenic organisms. If the pyogenic organism is first to establish itself, the secondary invasion with a mold is rare.

The filamentous forms are the chief offenders in producing otomycosis. They produce intense itching in the course of their growth, and cause epithelial exfoliation which fills the external auditory canal with mushy débris. Sometimes the white filamentous growth is visible and if sporulation has occurred, the black color of the myriad spore capsules gives the appearance of coal dust in the canal. Secondary invasion with pyogenic bacteria also occurs

in this type of infection and may make the treatment of the case more difficult.

Higher bacterial forms comprise the Actinomyces and the Nocardiae, both acid-fast and non-acid-fast; they are of relatively little importance because of their rarity.

Allergic reactions are seen as result of contact with some specific substance to which the patient is hypersensitive. Metal and composition spectacle bows and cosmetics may be mentioned as illustrations of local reactions to specific stimuli.

Eczematoid lesions in which vesiculation and serous exudation are prominent features may take place during the course of an otitis externa and give rise to a most distressing condition that is highly resistant to treatment. Ceruminous plugs sometimes irritate the canal and cause denuded areas which are easily infected. Vitamin deficiency or glandular insufficiency may account for the persistence of chronic inflammatory lesions of the pinna and external auditory canal.

The dermatoses such as dermatitis seborrhœica, herpes simplex, and herpes zoster as well as tinea infections must all be borne in mind in differential diagnosis as they produce lesions which may prove embarrassing if not properly diagnosed at the time.

In the differentiation of otitis externa the history as well as the objective findings will suffice to make a diagnosis in the majority of cases. Examination of a smear taken from the ear will reveal budding forms and filaments and makes distinction between them easy but does not suffice to classify exactly the responsible organism. Cultures are necessary for this purpose and the final classification of any mold depends upon a study of its fermentation reactions in the various sugar media as well as exact study of its morphologic characteristics. Petri dish culture gives important information upon which classification may be based. The most important and valuable method of culture which yields the greatest information in studying morphology of molds is the slide culture, where the suspected material is implanted in a culture medium and allowed to grow between two slightly separated slides.

Treatment of otitis externa consists essentially in first removing the irritant whether it be physical, chemical, or bacterial; limiting its action, and preventing a recurrence of the effect. The treatment of otitis externa due to thermal and mechanical causes as well as that due to radiant energy is obvious. Our principal concern is the treatment of the infectious group.

For treatment of the pyogenic types a great variety of drugs have been used, but the investigations of McBurney and Searcy[1] show that many of them are almost wholly without effect. It is imperative that pain be controlled by adequate sedation. The most valuable local application in our hands has been metacresyl acetate applied full strength on a cotton wick which is allowed to remain in situ twenty-four hours before being changed. It is usually necessary to reintroduce it at the end of twenty-four hours after cleansing the parts of all exfoliated epithelium. Metacresylacetate is anesthetic, bactericidal and fungicidal. It has been of great value for this reason, as control of itching and pain is just as essential to the patient's comfort as eradication of the infection. If metacresylacetate is not available, a very good substitute is camphor-phenol mixture, N.F., which may be used straight or with a small percentage of iodine, ½ to ¼ per cent; or even more may be added to it and its fungicidal properties somewhat enhanced. It is not quite as good an anesthetic as metacresylacetate. Both these preparations are liquid and may be applied on a cotton wick inserted into the depth of the auditory canal after adequate preliminary cleansing has been carried out. Bismuth violet, oil of cade in ointment form, X-ray therapy, vaccines, and staphylococcus bacteriophage, are all of service at some stage in treatment. Thymol is particularly valuable in combination with metacresylacetate or alone in oily solution or suspended in an inert powder. A combination of tricresol, iodine, and thymol 1 per cent in boric acid powder is valuable for insufflation into the canal if the patient cannot come for daily treatment.

The treatment of the mold infections is essentially the same as that of the pyogenic types except that iodine has a special affinity for the molds and is quite often of value in ointment form or in weak alcoholic solution applied to the infected parts. The use of potassium iodide internally has been recommended to limit the growth of molds.

The newer drugs of the sulfa class are of definite value in some of the pyogenic types, both when used internally and locally, but so far as their effect on molds is concerned, we have so far not been impressed with their effectiveness.

One should bear in mind that reinfection is frequent and adequate measures should be employed to guard against its taking place. Swimming pools particularly should be under the strictest hygienic supervision to prevent contamination. Local prophylactic measures should be available and if there are other areas of

infection on the body, pyogenic or mycotic, they should be treated simultaneously with the condition present in the ear.

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XXXV

AMELOBLASTOMA OF LEFT MAXILLARY SINUS

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D. P., an Italian male, aged 52 years, was referred to me for sinus examination on Nov. 8, 1938.

There was a history of his having had the left upper second molar extracted about twenty-five years ago by a barber, as a result of which he had a sore face and upper jaw for two or three weeks after the extraction, with a sinus tract into the left antrum that has never closed completely.

The present complaint goes back several years. At intervals of three or four years, when the patient had a cold he would have a discharge of dark bloody thick secretion through the second molar alveolar space for two or three weeks, after which it would heal. In June, 1938, a dentist had washed out the second molar cavity, and the sinus tract into his antrum closed in three weeks. About four months ago before the present examination he had had some ureteral calculi removed, and it was while looking for a primary focus of infection that his family doctor had his sinuses X-rayed and referred him to me for sinus examination and treatment.

It was difficult to convince this man that he needed sinus surgery of the left antrum, because he had no symptoms referable to this sinus. The only thing he noticed was, that every two or three years he would have a foul-tasting bloody discharge from his second molar tooth socket. He sometimes attributed the gum bleeding to chewing on the hard crust of Italian bread. At no time did he complain of pain in the left side of his face or of any discharge from his nose.

Examination by transillumination on Nov. 8, 1938, revealed that no light penetrated the left antrum. The right antrum showed a slight decrease in illumination, as did the frontals. There was no external swelling of the face nor any asymmetry on palpation of both cheeks. The nasal passages on both sides were clear, and there was no evidence of a new growth in the nose. There was however, a deep red congestion of the turbinate, but no turgescence. The

X-rays taken previously corresponded with the findings by transillumination.

There were several teeth missing from the upper jaw on the left side, and there was a sinus tract from the second molar alveolar socket communicating directly with the antrum. A Caldwell-Luc procedure was performed at the Victory Hospital on Nov. 12, 1938, and the patient was discharged the following day. The entire antrum was completely filled by a mass which was solidly impacted, homogeneous, smooth surfaced and dark red in color. This mass was removed piece by piece because its size and consistency would not permit of its passage through the operative opening in the canine fossa.

The immediate postoperative course was good. Temperature, pulse and respiration were within normal limits. The patient has remained symptom free. The window under the inferior turbinate is still patent and can be freely probed. The preoperative sinus tract is still present but there has been no discharge from this. He wears a small prosthesis to cover an opening the size of a buckshot.

Urinanalysis showed an alkaline reaction, a specific gravity of 1.012. The acetone was negative, diacetic acid was negative, the albumin was negative, the glucose was negative; there was no evidence of epithelial cells, no cylindroids but there were a few shreads and bacteria. The blood picture showed 4,600,300 red cells and 6,800 white cells, a hemoglobin of 90 per cent, a color index of 0.9; the white cell differential count showed 70 polymorphonuclears, 28 small lymphocytes, and 2 monocytes. Blood pressure was 118/84. The pathologic report was as follows: "The microscopic section shows closely packed adenomatous structure. Pathological diagnosis: adenomatous polyps from the antrum." The X-ray report was as follows: "the membranes of all the sinuses show some thickening but the left antrum shows a very definite clouding of the entire structure."

The patient has gained weight since the operation and he has not varied in weight in years. He feels constitutionally better. The foul taste and odor in his mouth have disappeared.

Of course, he will be under observation from time to time for the next five years, to observe whether or not there is a recurrence, and if so a biopsy will be taken to determine whether the microscopic picture is the same. It is now three and one-half years since operation and there is no evidence of recurrence.

The unusual feature of the pathology in this case was brought to my attention by the pathologist at the hospital. For this reason a specimen of the tissue was forwarded to Colonel Ash of the Army Medical Museum, Washington, D. C., for further observation, examination and diagnosis.

His report is as follows: (A.M.M. Acc. 75597) "*Gross:* four bits of tissue, the largest measuring 1.5 by 0.5 cm. The smallest measure 3 by 2 mm. All four pieces are similar in appearance, being of a light yellowish color, smooth surface, and on cut section have the consistency of an olive and are of a quite homogeneous appearance.

"*Microscopic:* The section reveals several pieces of tumor tissue covered by a fairly normal layer of respiratory epithelium. The stroma is essentially polypoid in character, loose and edematous and the cells have a stellate appearance. Throughout this tissue numerous islands of epithelial cells are noted. Some areas of these collections are dense, closely packed and, in others, more loosely arranged. Some of the cell collections show varying stages of central degeneration. Under higher power the similarity of the outer cell border to the inner enamel epithelium of the tooth bud is easily recognized. Masson stains showed these collections to be entirely epithelial.

"*Diagnosis:* Ameloblastoma, maxillary sinus."

Robinson[1] says that "the ameloblastoma may be defined as an epithelial tumor arising from odontogenic apparatus or from cells with a potentiality of forming tissues of the enamel organ. Its structure resembles histologically that seen in various developmental stages of the enamel organ. Thoma[2] preferring the term adamantoblastoma says: "The adamantoblastoma is derived from epithelium that has the power to differentiate into enamel epithelium without actually forming enamel." Thoma points out, moreover, that this tumor has been called an adamantinoma; however, since it is not made up of enamel but consists wholly of enamel-organ type of epithelium, the term adamantoblastoma describes the histologic makeup much better. Churchill prefers the term ameloblastoma which conveys the same idea, although the ameloblastic stage of development is not reached in every instance. The term ameloblastoma is derived by combining "ameloblast" designating the enamel-forming cell, with "blastoma", designating a tumor of one type of tissue. In addition, there are at least twenty additional synonyms in the English language and a large number in the foreign languages.

The term adamantoma has been accepted by some to describe the corresponding hypophyseal tumor.

According to Thoma[2] the tumor may form from cells that have already become differentiated or from embryonic epithelial cells. In the latter instance the result will vary according to the stage to which the cells differentiate during the tumor development. It must be remembered that no matter from what structure the tumor has formed, it inherits the potentialities of the embryonic oral epithelium, which through down-growth develops both adamantoblastic and glandular structures, and that therefore, acini of glands occasionally may be found in the tumor.

The tumor may be derived from (1) genetic disturbance of the enamel organ or the tissue forming it, (2) cell rests of the enamel organ, either during tooth development or later, (3) the epithelium of odontogenic cysts, (4) the surface epithelium covering the jaw, and (5) displaced epithelium in other parts of the body.

From the earliest reports it has been considered that the ameloblastoma is in some way derived from some part of the odontogenic apparatus or from cells with a potentiality for forming the odontogenic tissues. Robinson[1] lists the essentials of the various theories of etiology in tabulated form as follows:

1. The ameloblastoma takes origin from the odontogenic apparatus:
   - (a) From overgrowth of the buds or from excess of buds
   - (b) From the oral epithelium
   - (c) From epithelial débris
   - (d) Indirectly through dentigerous cysts
2. Stimulation for the growth may result from:
   - (a) Irritation
   - (b) Dietary disturbance
3. The ameloblastoma is a form of:
   - (a) Squamous-cell carcinoma
   - (b) Basal-cell carcinoma.

It seems reasonably certain that the primordium for this neoplasm is some part of the odontogenic apparatus or cells with a tendency to form this apparatus. This conclusion is based chiefly on the morphologic characteristics. The periods of formation of the anlage of normal tissue and of associated embryonic rests have been given by Li and Yang, who placed a period of origin of the enamel organs and of the ameloblastoma in the sixth week of embryonic life. Whether this primordium is actually an excess of tooth buds, an overgrowth of an individual bud, oral epithelium or epithelial débris in any of its forms has not been demonstrated. The theory that irritation has incited the primordium to proliferate does not appear to be ungrounded. A summary of the cases recorded in the literature shows that 44 cases were preceded by extraction of teeth, 21 were preceded by oral infection and 14 cases were associated with unerupted teeth contained in the mass.

As many of the histories in these cases suggest a traumatic or irritating factor in the form of surgical disturbance, injury, infection, impaction or difficulty of eruption as well as an oral disturbance in some instances, it seems that there may be a primordium of cells with a tendency to form the epithelial portion of the tooth germ, which may be excited to growth by some irritating secondary factor.

Churchill's classification in 1932 of ameloblastoma as a dentocystic tumor derived from the odontogenic apparatus of the squamocellular mucosa seems to be the most adequate of those encountered. Likewise his subdivision into benign ameloblastoma without or with cornification in solid, cystic or melanotic form and the malignant sarcoma ameloblasticum and carcinoma ameloblasticum covers most of the types that have been described. The term "adamantinoma" is suggestive of a hard tumor of enamel, and the descriptive name "ameloblastoma," proposed by Ivy and Churchill (1929) is preferable for this epithelial growth.

Robinson[1] gives carefully compiled data arranged in tabular form of 379 cases of ameloblastoma studied from the literature. Of these 379 cases, 142 were male and 169 females. The average age at the discovery of the tumor was 30 years. It is important to note that the site of the tumor in 247 cases was the mandible and in 48 cases, or 16.3 per cent, the maxilla. In the maxilla, the maxillary sinus generally becomes involved with expansion of both the facial and nasal walls. As the tumor often exists for a long time before the patient has any knowledge of it, age statistics are very deceiving. He also reports that the structural changes in 219 cases were as follows: cystic, 124; cystic and solid, 53; and solid, 42.

The ameloblastoma is usually a central tumor, but as has been pointed out, there are cases on record which are of peripheral origin. They may be solid or cystic. In some cases the tumor is contained in a single compartment, when it simulates an odontogenic cyst. It may even contain a tooth so that the diagnosis of dentigerous cyst seems logical and the mistake generally is found only at the operation.

Teeth are quite frequently found in ameloblastomas. More frequently the adamantoblastoma is of the polycystic type giving the appearance of a multilocular cyst. The development is by extension and the formation of additional cysts by invading epithelium. The tumor may cause a swelling of the alveolus or of the entire jaw. If the bone is completely resorbed the cyst may be fluctuant but not painful on palpation; the soft tissue, however, is not invaded except in malignancy. Ameloblastoma has been reported in many cases in the hypophysis, and in at least seven cases in the tibia. Robinson found that the ratio of occurrence between the black and white races to be 11:1. Twice in the series the ameloblastoma occurred atypically about the face, once in the upper lip and once in the orbit.

Most of these ameloblastomas were in cystic form at the time of operation, 57.5 per cent of the 219 instances in which this feature was reported being cystic, 24.2 per cent both cystic and solid, and 19.1 per cent solid; 14 of the reported tumors contained unerupted teeth. To these ameloblastomas containing teeth Kaufman (cited by Thoma) has given the name "odonto-adamantinoma". Most authorities are agreed that the ameloblastoma never forms enamel.

The question of malignancy is discussed by all writers and is a matter of great importance. It has been stressed that most adamantoblastomas grow slowly and distend the jaw more than they destroy it. The malignancy of these tumors has long been in question. Anatomically they are usually benign, but a few scattered ones have been reported which were histologically suggestive of malignant growth. Likewise in some instances, metastases have been reported; a few resulted in death by local extension, and evidence suggesting metaplasia, carcinoma and sarcoma has been seen in others.

There are 17 cases among those reviewed in which there seems to have been given some evidences of malignancy. However, the common metastases have been reported, even among the cases in the following list, without histologic examination of the metastatic

growth. Some of the reported metastases have not been submitted to even gross pathologic examination. In nine cases metastases appear to have been observed in the presence of ameloblastoma, and secondary growth probably was ameloblastomatic in nature. Seven tumors showed histologic pictures of a malignant type, according to the writers who reported them. From the descriptions given they have been classified as carcinoma ameloblasticum and sarcoma ameloblasticum. Metaplasia to more malignant growth has been observed in four cases cited from literature.

Since it is not felt that metaplastic growths, local extensions or implantations should be considered as evidences of malignant growth, only the aforementioned nine cases of metastasis, seven cases of histologically malignant ameloblastoma, and one other case can be accepted as instances of possibly malignant growth. The 17 tumors listed here represent only 4.5 per cent of the 379 ameloblastomas surveyed. In view of the foregoing findings it appears that ameloblastomas are benign and the relatively small number of malignant ones are atypical.

Local metastases according to Thoma[2] occur very rarely; sometimes they are due to the adamantoblastic epithelium; at other times to sarcomatous changes in the stroma of the neoplasm. Such tumors are spoken of as adamantosarcoma. In other instances the metastases may be due to transformation of the tumor into epidermoid carcinoma or adamantocarcinoma.

Metastases to the lymph glands have been reported, but only in a very few cases. It must be kept in mind that enlarged lymph glands, especially submaxillary glands, may be enlarged on account of coexisting infection of teeth or the mucous membrane in the mouth. Excision of the gland and microscopic examination, therefore, are necessary to make a diagnosis of tumorous involvement. Metastases to the lungs have been described especially in patients undergoing operation under general anesthesia and particularly where the antrum is involved and the growth impinges upon the nasopharynx.

Recurrence is common and is due to the fact that because the tumors grow slowly, the surgeon is tempted to be too conservative. It must be kept in mind that conservative treatment with incomplete removal tends to disrupt the expansive growth of the tumor and induces a more malignant and infiltrating type which may involve inaccessible and ultimately fatal locations. The recurrence is due to proliferation of epithelium in the spongiosa or the haversian sys-

tem of the cortex. This infiltrating character is more common in the undifferentiated type. Infiltration of the soft tissue is not a common characteristic. In Robinson's survey there were 119 cases in which from one to 22 recurrences were reported.

Gross examination discloses two main types, the monocystic which contains a solid tumor mass and the polycystic type in which small or large cysts that may contain fluid occur; but the cystic type may become solid by ingrowth of tumor tissue from the wall, while the solid type may become cystic through changes occurring in the follicles. The solid tumor is usually white in color, of fine granular consistency and encapsulated. In the cystic type we find some compartments filled with solid tumor and others which are cavities lined by a smooth membrane consisting of epithelium backed by a small amount of connective tissue. In such cysts there is a clear, yellow fluid, sometimes of mucus, sometimes of colloid material. The expanding bone may become very thin or may be resorbed completely, but the periosteum generally is not involved and osteoblastic changes do not occur.

Microscopic investigation shows that the tumor is subject to many variations due to the stage of differentiation; the epithelium may attain the behavior of the stroma, and malignant changes may develop. Pure forms are seldom found. A number of histologic types are recognized; for instance, epithelioma type of adamantoblastoma, stellate type of adamantoblastoma, adamantinocarcinoma, melanotic adamantoblastoma. The ameloblastic type of adamantoblastoma forms lobes or follicles made up of cells such as are found in the third stage of development of the tooth germ. At the periphery the cells are cylindrical, reaching the ameloblastic shape. They are backed by a layer of indifferent cells followed by stellate ones in the center.

For a complete and full account of the histology of ameloblastoma the reader is referred to Robinson.[3]

Careful X-ray examination and study is recommended to find out whether it is possible to avoid resection. A biopsy examination before treatment is recommended. It not only serves to differentiate between cysts and adamantoblastoma and other central tumors, but also helps to detect malignant tendencies in both the tumor epithelium and the connective tissue stroma. The consensus of opinion seems to be that a fairly radical excision performed when the tumor is small may save the patient recurrences and repeated and more serious operations. Conservative operation may be advised

for young people with small tumors, if the patient agrees to frequent examination and subsequent radical operation if there is recurrence. For extensive tumors resection far away from the lesion is advised to prevent recurrence, but with such resection there is always the possibility that the patient may succumb to resulting cellulitis or bronchial pneumonia. To avoid radical resection, irradiation treatment has been advised after more conservative excision, particularly the use of radium after surgical removal and periodic control with X-rays. Surgical diathermy has also been used as a treatment of choice.

While the ordinary ameloblastoma is a benign, slowly growing tumor it should always be remembered that a slowly growing tumor may become malignant and grow more rapidly and that a clinically and even roentgenographically typical adamantoblastoma may change after years into adamantocarcinoma or adamantosarcoma.

The most important problem the surgeon is confronted with is whether a particular tumor is benign or malignant. Thoma quotes Simmons who states that in his opinion the cases in which the cuboidal cells predominate present probably the more malignant form of growth, while in cases of the cystic type recurrence is slow and can easily be recognized. Any growth in which the rate of growth is suddenly increased should be given a guarded prognosis. It is best, of course, that a biopsy examination be made to determine the type of treatment for each case.

The ameloblastoma should be treated as an expansive benign growth with a tendency to recur. In many cases the recurrence can be traced to incomplete enucleation of the tumor from the numerous crypts and lacunae within the bone. These findings agree with Ewing's statement that "in spite of their relative benign structure, the prognosis in this group is unfavorable." They are persistent, not malignant, tumors.

SUMMARY

According to Robinson the ameloblastoma is usually nonfunctional, intermittent in growth, anatomically benign and clinically persistent. According to the same author the first fact that becomes evident to a reviewer of the literature of the subject is that the ameloblastomas are not as rare as is generally believed.

Most of the growths are cystic at the time of operation. Some are cystic and solid and the smallest number are solid. The malig-

nant cases represent only 4.5 per cent of the total number of instances of ameloblastoma collected. While ameloblastomas are not typically malignant, their prognosis is unfavorable because of the difficulty of complete removal and their persistent nature.

Etiologic considerations are at best based only on theoretical grounds, in view of the present inadequate knowledge of the dentocystic tumors. It does seem that the ameloblastomas rise from some part of the odontogenic apparatus or cells with a potentiality for development into tooth-forming tissues. This primordium may be incited to proliferate by irritation from retained teeth, surgical operations on the teeth or jaws, injury, infection or possibly general disturbances of the economy.

The ameloblastoma never forms enamel and is not an extremely hard tumor. The term "adamantinoma" is therefore a misnomer. Of the cases of ameloblastoma collected, 83.7 per cent occurred in the mandible. Irritation in the form of retained teeth, infection, injury, operative trauma or general disturbance seems to be an etiologic factor, stimulating a primordium with a potentiality for the formation of the enamel organ.

CONCLUSIONS

1. A case of ameloblastoma of the left maxillary antrum is reported. The case was originally reported as an adenoma of the antrum before the Celtic Medical Society at the December, 1938, meeting.

2. The true nature of the pathology in this case was realized after receiving Colonel Ash's letter from the Army Medical Museum in Washington, D. C., at which place the tumor has been registered under Otolaryngological Pathology. The consultant committee agreed universally on the diagnosis.

3. A résumé of the outstanding contributions of this subject by Robinson and Thoma has been given.

4. This tumor is of odontogenic origin. It is definitely a borderline case. From a surgical standpoint it has its origin in the field of dental or oral surgery but its removal comes within the province of the rhinologist, i.e., its origin is dental and the surgical treatment is rhinological.

5. The patient is well, symptom free, and shows no signs of recurrence after three and one-half years. He has not been subjected

to any radiation therapy. He is still reporting for routine check-up every two months. If there should be a recurrence, a biopsy will be taken for microscopic examination and treated further accordingly.

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XXXVI

UPPER RESPIRATORY INFECTION OF A FULMINATING CHARACTER REQUIRING TRACHEOTOMY*†

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In this type of infection we are confronted with a very severe disease which has to do with the airway. Many times we have a more or less moribund patient who must have an artificial airway made immediately after being admitted to the hospital in order that his life may be saved. In the discussion of this condition the following points will be taken into consideration: bacteriology, macroscopical and microscopical pathology, symptomatology, diagnosis, complications and treatment. The discussion is based on 79 cases of emergency tracheotomies which were done at the Indiana University Hospitals, 16 of which were due to this type of infection.

A number of investigators are considering a virus as the primary invader, but as yet they have not found enough evidence to establish this hypothesis. It is not beyond reason to think that this is true and that symptoms which are manifested are due to secondary invaders such as the streptococcus haemolyticus and Streptococcus viridans, these being the most common invaders, although at times the condition may be due to other invaders such as the pneumococcus, Micrococcus catarrhalis or staphylococcus; however, the latter do not produce as severe a symptomatology as the first two mentioned. Whether or not the above-mentioned organisms are primary or secondary invaders following a virus infection, the symptomatology is practically the same.

The macroscopic pathologic picture as is evidenced by observation is of importance. It reveals that the mucosa is red, velvety and swollen, and the rings of the trachea and bronchi are obliterated. The mucosa may be oozing blood in areas and there may be crusts or plugs of mucus in the bronchi.

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†Presented before the section meeting of the American Laryngological, Rhinological and Otological Society, St. Louis, Jan. 21, 1942.

A very deep red and slightly edematous larynx is quite characteristic. It is known that the subglottic areas have a tendency to spread mesially due to the character of the connective tissue, especially in children where it is very loose.

The microscopic pathology shows an inflammatory change in the mucosa and submucosa. The swelling produced by this inflammatory change occludes the smaller bronchi and may produce death. The secretions of the endobronchial tree evidence a pathologic change which is very viscid. They lose their fluid character and subsequently crusts and plugs are formed. This condition may be aggravated by certain drugs, especially morphine and atropine, or by the lack of fluid intake and poor atmospheric conditions.

The symptomatology which is presented in this condition is well known to all of us, but it should be enumerated in order that the whole picture may be carried through. This fulminating type, with fever and oftentimes a chill, is followed by toxemia and laryngeal and bronchial obstruction. Cyanosis may be present, but there is likely to be an ashen gray color even when asphyxia is present. The suprasternal notch, the clavicular spaces, the epigastrium, and the intercostal spaces are indrawn. There is considerable wheezing and stridor with a croupy type of cough.

There is more dyspnea on inspiration, although this may occur upon both inspiration and expiration. We must of necessity recognize the laryngeal and bronchial obstruction early, and unless it is dealt with promptly and in a radical manner, the patient's demise is by asphyxia or atelectasis, or, as Jackson so aptly puts it, he "drowns in his own secretions". Cough may be absent or may occur at the beginning and then disappear.

In making a diagnosis all physical signs should be taken into consideration first, but the evaluation of these signs may well be left until roentgenograms of the chest are made. If necessary, a bronchoscopic examination should be made. By so doing, a great deal of the field involved is brought into view. The type of secretion should be noted and the bronchi should be observed for plugging. No doubt a differential diagnosis can be made more rapidly with a bronchoscope than by any other manipulation.

Any of the complications of the upper respiratory tract following acute infection may occur; i. e., blood stream infection, or cardiac involvement which may be fatal, such as myocarditis or endocarditis. Exhaustion and inability to breathe may be mislead-

ing. These pseudopneumonic signs may suggest pneumonia, but we are pleased to find that what appeared to be a pneumonic process is only a bronchial obstruction due to crusts or plugs.

The patient should be hospitalized and should be under the supervision of a medical attendant, and in order that an evaluation of all studies may be properly interpreted, proper feeding should be maintained both for cardiac and pulmonary conditions. The patient should not be worn out by undue attention. Probably the most vital thing to the patient is the maintenance of the airway, and this duty is placed upon the laryngologist.

In the treatment of this type of infection, the first procedure to be carried out is to have the patient breathe properly humidified air. This is best secured by the use of one or more of the new cold-humidifying apparatuses. By this method, the temperature of the air is not elevated and the room temperature can be maintained at an optimal 72 degrees. The amount of humidity desired is 70 per cent, measured on a wet and dry bulb hygrometer. The maximum and minimum limits of humidity are 80 per cent and 60 per cent, respectively. The nurse reads and records the hygrometer readings hourly. By having the room properly humidified, it has been found in the Indiana University Hospitals that crusting of the tracheobronchial tree due to inspissated pus and mucus has been reduced to a negligible amount. It may thus be seen that this is probably one of the most important factors in the care of these patients.

It has been found in this institution that more than one cold humidifier is necessary when the temperature outside the patient's room is below freezing. It must be said at this point, also, that proper ventilation from the outside must be maintained at all times to make sure of a normal oxygen tension in the air. When the weather is cold outside, the vapor thrown by the humidifier is condensed rapidly on the outer walls of the room, and on the windows, thus rendering the machine less efficient. In hospitals where cold humidification is not available, steam may be obtained from a steam-heating system by opening a valve on the radiator or pipe in the room. The disadvantage of this method is that the room becomes overheated. When the attendant opens the window, it causes drafts and great fluctuations in the humidity level.

Patients may be grouped in a room somewhat larger than the ordinary hospital room and several humidifiers used, thus saving

both space and nursing personnel. The cold-humidification units must be kept on a small table or standard so that the generated vapor will be thrown high in the room, as the vapor settles to the floor rapidly. If the units are placed on the floor, humidity at the level of the patient will fluctuate with the drafts in the room. Strict instructions are issued to the nurses and employees, so that there is no useless opening of the room door.

If the patient is not sufficiently relieved in his respirations by this procedure, a tracheotomy is then necessary. This should be done early when it can be done deliberately, and all the disturbances should be avoided which go with an emergency procedure.

The tracheotomy should be done by using infiltration anesthesia, preferably adrenalin and novocain, and the incision should be low. The great advantage of tracheotomy should encourage an early resort to this procedure instead of postponing it until the patient is in extremis as is frequently done.

Following tracheotomy, two types of dressings for the fresh wound are used. The first consists simply of a dry gauze dressing of the type commonly termed "flats" and so cut that they fit snugly about the tube. After the operation, this dressing is not disturbed for 48 hours, at which time the tube is changed and a clean dressing applied. The second method differs only in that the dressing is kept saturated constantly with bichloride of mercury solution 1:5000. After the 48-hour period following tracheotomy, the attending physician then changes the tube one or two times daily at his own discretion, governed, of course, by the amount and tenacity of the patient's secretions.

From the time the tracheotomy is done until the patient is entirely out of imminent danger either from suffocation by drowning in his own mucus or pus or from crusting of the tracheobronchial tree, a nurse is kept on constant 24-hour attendance. It is a maxim in the University Hospitals that the child newly tracheotomized shall not draw an unwatched breath for five days. As a general rule, a more severe case will require more than five days of special nursing care.

The nursing duties are outlined as follows: The nurse is to make rather voluminous bedside notes; to keep charts of the patient's color, temperature, respiratory rate, and pulse; and to record, as mentioned before, the humidity and temperature of the room.

If the patient shows any signs that arouse anxiety in the nurse, she is told to feel free to call the physician at any time. Her other duties include the moistening of the dressings when required. Most important, she must perform frequent aspiration of the outer cannula and cleansing of the inner cannula. Aspiration of the outer cannula is done in the hospital with a No. 10 or No. 12 soft rubber catheter with at least three holes near the tip so that by no means can the mucous membrane of the trachea be pulled up by the suction. A small piece of adhesive tape is placed about two or three inches from the tip, according to the length of the tracheotomy tube. This is done so that the nurse will not pass the tube into the trachea or bronchi at the time the inner cannula is removed for aspiration of the outer cannula.

It is oftentimes necessary to resort to the bronchoscope in the removal of mucus, mucous plugs, and crusts when these cannot be removed by the above-mentioned procedure. This is done by passing the scope through the tracheotomy wound. Occasionally this procedure has to be repeated many times, and in one case it was done over thirty times. The inner cannula is cleansed by pulling through the tube a piece of tonsil snare wire containing in its loop a small piece of soft gauze. The nurse is also instructed to see that the surgical tapes encircling the patient's neck are at a proper tension. Of course, the nurse has the general care of the patient in addition to these special duties. She must chart the fluid intake and output and give the patient routine bedside care.

One additional alteration of the inspired air is sometimes necessary by the administration of oxygen. The requirements for the use of oxygen are cyanosis in the presence of an adequate airway, a rapid respiratory rate, or a rapid pulse rate. If the patient has an adequate airway, it has been found that cyanosis is more frequently due to general toxic effect, while elevation in the respiratory rate without cyanosis is more often due to concomitant bronchopneumonia. Oxygen is administered by means of a funnel tied so that it is suspended over the opening of the tracheotomy tube and about one inch above it. The oxygen delivered through the funnel is first humidified by allowing it to pass through water. It is then felt that oxygen, as it comes from the tank, is extremely dry and in itself will cause crusting of the tracheobronchial tree.

It has been found in some cases that for a few hours immediately following tracheotomy there is a partial and sometimes momen-

tarily complete apnea. This is due to the gradual elevation of the carbon dioxide threshold prior to the establishment of an adequate airway by tracheotomy. When the tracheotomy is performed, the patient quickly washes out the carbon dioxide and because of the altered threshold the respiratory center is temporarily not stimulated. At times this is seen to be a severe phenomenon, and artificial respiration may be required immediately following tracheotomy. It has been stated that patients have died because of this alteration in the normal physiology. If the administration of carbon dioxide is required over a long period of time, it is given by the same method as is described before the administration of oxygen; also if it is to be continued for some time it is mixed with oxygen.

More recently we have employed the technic in treating these patients described by F. W. Davidson.[1]

If difficulty is caused by the thickness and tenacity of the tracheobronchial secretions, it is combated by the use of intratracheal drops. These drops consist of normal saline solution with ten drops of adrenalin 1:1000 per ounce. Five to eight drops of this solution are placed in the tracheotomy tube as often as necessary. By this method the patient draws the solution into his trachea and bronchi and it is immediately aspirated by the attendant. Frequently tablets of calcium iodide are given to convalescing patients by mouth and the same result is achieved. In the general care of these patients, a high fluid intake is maintained and given intravenously when necessary. The diet is given as tolerated. No sedatives are given at any time.

The use of sulfonamide preparations in the treatment of this type of case has been, on the whole, unsuccessful. It has been given both before and after tracheotomy, but the usual picture of rapid resolution in temperature and relief of toxic symptoms has not resulted. It is still felt in a great many cases, though more from hope than past experience, that some benefit may be derived from sulfonamide therapy and, as a result, many patients receive it.

As soon as the tracheobronchial secretions are diminished sufficiently and there is no embarrassment of respirations, the tube is corked. A one-quarter cork is usually inserted first and if tolerated for 24 to 72 hours, it is increased to a one-half cord and so on through the three-quarter size until a full cork has remained in place for 24 to 72 hours and the child is in absolutely no respiratory difficulty. The child is then decannulated.

SUMMARY

1. We have to deal with a very grave type of illness.

2. The bacteriology should always be given consideration, both in diagnosis and treatment.

3. The macroscopic pathologic picture often has to do with the type of treatment employed.

4. Treatment should be directed toward improving the airway. If possible, it should be palliative, but if severe symptomatology is present, then we should resort to tracheotomy.

5. Tracheotomy should be low and done early.

6. The posttracheotomy treatment should be done with meticulous care, and the patient should be under constant observation, the airway being kept free of viscid mucus and crusts.

7. The patient should be decannulated gradually, and the tracheotomy tube should not be removed for from 24 to 72 hours after the tube has been completely closed.

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XXXVII

THE EFFECT ON HEARING OF EXPERIMENTAL OCCLUSION OF THE EUSTACHIAN TUBE IN MAN*†

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BALTIMORE

Two centuries ago Valsalva reported that a patient with a pharyngeal ulcer had temporary impairment of hearing when, in the course of treatment, the eustachian tube was closed by an applicator. Since his time it has been recognized that any interference with the normal functioning of the eustachian tube causes impaired hearing. The usual idea has been that tubal occlusion impairs chiefly the perception of low tones. Bezold, Siebenmann, Struycken, and others of the earlier authors realized that impairment of hearing for high tones can occur in catarrhal affections of the middle ear. They did not state definitely the location of the causative lesions, and most otologists have regarded poor hearing for high tones in patients with tubal occlusion as proof of involvement of the inner ear. Crowe and Guild, however, presented evidence that the impairment caused by adenoid obstruction of the tubes begins with the high tones. Because the hearing frequently returns to normal after restoration of good ventilation of the middle ear, these authors believe that poor hearing for high tones can be caused by changes in the middle ear too slight to interfere with the transmission of low tones.

However, in patients with tubal occlusion the impairment of hearing may be the resultant of several factors, and the possibility cannot be ruled out that one of the factors is a reversible condition of the inner ear. Among the plausible suggestions that have been made are edema of the organ of Corti (Mygind) and influence of toxic substances absorbed from the middle ear (Galloway). Only by acute experiments of short duration can such possibilities be ruled out and the primary effect on hearing of the occlusion itself be determined. It is difficult to interpret, in terms of clinical observations, the few reports of animal experiments that have been made, and no one seems to have used man for such experiments. The latter fact is

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†Aided by a grant from the John and Mary R. Markle Foundation.

surprising, in view of the well-recognized importance of the eustachian tube for normal functioning of the middle ear and of the simplicity of the technical procedures necessary.

Acute occlusion of both eustachian tubes was obtained experimentally by inflation of a small rubber balloon in the nasopharynx. The collapsed balloon, the mouth of which was glued to a soft rubber catheter, was inserted through one side of the nose and inflated with air until breathing through either side of the nose was completely blocked. Anesthesia was obtained by spraying the nasopharynx and one side of the nose with 1 per cent butyn in a 1 per cent aqueous solution of ephedrine and application of one drop of 20 per cent cocaine. Hearing tests were made before and after application of the local anesthetic, immediately after inflation of the balloon, at intervals of about fifteen minutes for the next one and one-half hours, and again after deflation and removal of the balloon, as soon as ventilation of the middle ears had been re-established by the positive Valsalva procedure and swallowing.

The occlusion experiments were made on the author only, because he hesitated to subject others to the possibility of complications, such as congestion, infection, etc. For the control experiments on the effects of local anesthetics and on normal variations with time, several other persons were also used.

Three occlusion experiments were made, the first two separated by a month, the second two by six months. The long interval between the second and third experiments was necessitated by an upper respiratory infection that began soon after the second experiment. Each experiment was followed by discomfort and a sensation of congestion in the nasopharynx and of fullness in the ears for a couple of days.

The changes in hearing were similar in each experiment. Because occlusion was maintained somewhat longer in the third than in the first two experiments, the data from it are used for the illustrations.

Figs. 1 and 3 show, for the right and left ears, respectively, the thresholds of the observer at the beginning of the experiment and the range of the changes that occurred during the period of occlusion of the eustachian tubes. The changes at each time interval are shown in Figs. 2 and 4, in each subdivision of which the original audiogram is plotted as a straight line (zero of the chart) and the changes as differences from the original hearing. Downward on these charts means impairment of threshold.

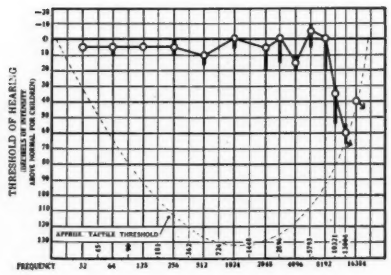

Fig. 1.—Right ear: original hearing and range of changes observed (vertical bars) during experimental occlusion of the eustachian tube.

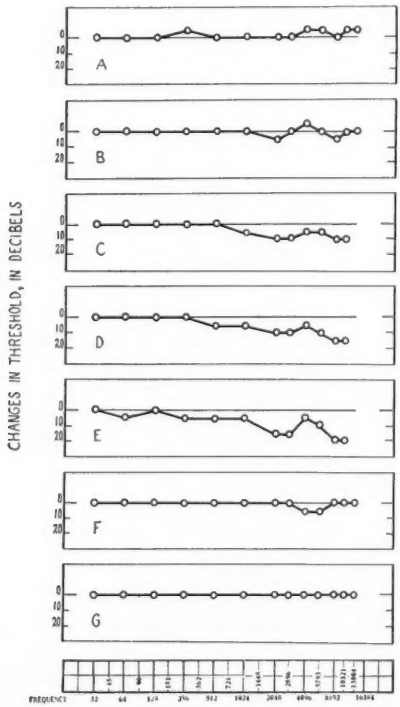

Fig. 2.—Right ear: changes of the hearing at different time intervals, compared with the original hearing (zero line). *A*, 10 minutes after beginning of the occlusion; *B*, 20 minutes; *C*, 45 minutes; *D*, 60 minutes; *E*, 75 minutes. *F*, after removal of the occluding balloon and ventilation of the middle ear. *G*, after a second ventilation.

Inspection of Figs. 1 and 2 (right ear) and of Figs. 3 and 4 (left ear) shows that the changes of threshold are greater for the high tones than for the other tones, that hearing for the high tones is affected sooner than for the other tones, and that immediately after restoration of adequate ventilation of the middle ear the thresholds return to the original readings.

Immediately after occlusion of the eustachian tubes was established a slight improvement in thresholds occurred for several frequencies, mostly high ones (*A*, Figs. 2 and 4). Within forty-five minutes (*C*, Figs 2 and 4) there was impairment of thresholds for high tones. The impairment increased with time and also involved additional, lower, tones. The transient improvements probably resulted from a temporary change in middle-ear pressure caused mechanically by the expansion of the balloon in the nasopharynx. The progressive impairments that followed were without doubt caused by the negative pressures developed in the middle ear by resorption of the contained air. The amount of negative pressure developed is not known, but certainly it increased during the period of occlusion. The fact that the hearing thresholds returned to the original levels immediately after adequate ventilation of the middle ear (*G*, Fig. 2; *F*, Fig. 4) is good evidence that in these experiments the hearing changes were caused by the altered pressure in the middle ear, not by secondary changes such as mucosal edema, hyperemia, fluid accumulation, etc.

Because the observer did not hear the highest tone (16,384 cycles) produced by the audiometer at the maximum intensity it generates (40 db.), the effect of the occlusion on this frequency could not be determined. For the frequency of 13,004 cycles the observations are also limited by the fact that the observer's thresholds were so near the limits of the audiometer (60 db. for this frequency) that the full extent of the impairments caused experimentally could not be measured. In Figs. 1 and 3 this fact is indicated by the arrows at the lower ends of the bars showing range of change. The impairment caused for this frequency was at least 20 db. for the left ear.

The changes in acuity of hearing for the frequency of 4,096 cycles are of special interest, because this is the frequency at which "dips" occur most often. In the present experiments the maximum changes in threshold for 4,096 in either ear were only 5 db., which is less than for the adjacent frequencies. In both ears the first change in hearing for 4,096 cycles was an improvement, and in the left ear this 5 db. improvement persisted after the hearing for adjacent

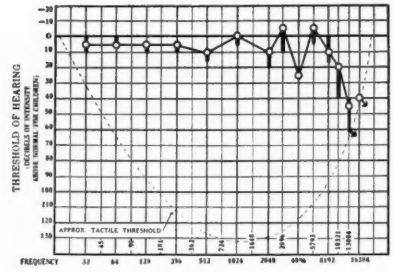

Fig. 3.—Left ear: original hearing and range of changes observed (vertical bars) during experimental occlusion of the eustachian tube.

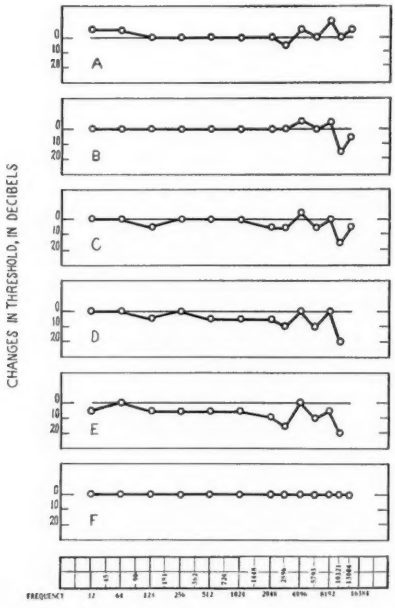

Fig. 4.—Left ear: changes of hearing at different time intervals, compared with the original hearing (zero line). *A*, 10 minutes after beginning of the occlusion; *B*, 20 minutes; *C*, 45 minutes; *D*, 60 minutes; *E*, 75 minutes. *F*, after removal of the occluding balloon and ventilation of the middle ear.

frequencies showed impairment (*C*, Fig. 4). Later, (*E*, Fig. 4) the threshold for 4,096 cycles was still at the original value when there was an impairment of 15 db. for the frequency a half-octave lower and of 10 db. for the frequency a half-octave higher. No satisfactory explanation has been found for the differential effect on the hearing of the frequency of 4,096 cycles, as compared to other high tones.

Although the changes for frequencies below 2,048 cycles fall within the range usually allowed for "normal variation" in repeated examinations of patients, in the present experiments these small changes do seem to be definitely significant because they occurred with an experienced observer, were always in the same direction, developed during the course of the experimental occlusion, and disappeared promptly after restoration of ventilation of the middle ears.

Control observations of three types were made: (1) on the spontaneous changes in hearing, (2) on the effects of the local anesthetics used in the occlusion experiments, and (3) on the effects of the pressure changes that could be caused without experimental occlusion.

Repeated hearing tests over a period of six hours were made on several observers, each of whom was free of clinical evidence of tubal or middle-ear lesions. For all observers more spontaneous changes occurred for high than for other tones, but they showed no regularity of pattern, either on comparison of the records of the different observers or of the records of the same observer on different days. Therefore the impairments observed during experimental occlusion of the eustachian tubes cannot be explained as spontaneous changes or "normal variation".

To test the effects on hearing of the local anesthetics used, repeated observations were made on several individuals after application of the amounts used in the occlusion experiments, and on himself the author also tried much larger doses (Fig. 5). In all cases the changes in hearing thresholds were small and did not show the consistent pattern found after occlusion. Since in these control experiments improvements of hearing occurred more often than impairments the changes in the occlusion experiments cannot have been caused by the anesthetics.

The third group of control observations was made in an attempt to evaluate the idea that the changes in hearing found in the other control experiments were caused by the small and transient changes

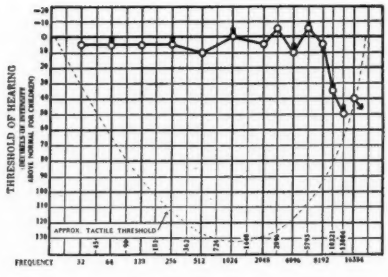

Fig. 5.—Toxicity control: original hearing and range of changes observed after application of butyn-ephedrine-cocaine in about four times the amount used in the occlusion experiments. Observation time, six hours.

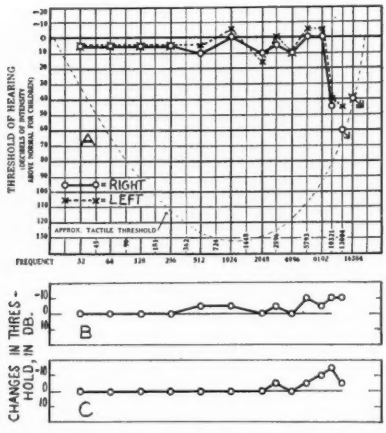

Fig. 6.—Hearing changes after application of ephedrine to the pharyngeal orifice of the eustachian tube and after ventilation of the middle ear. *A*, original hearing; *B*, changes after 15 minutes, compared to original readings (zero line); *C*, changes after positive Valsalva procedure and swallowing, compared to original hearing (zero line).

in middle-ear pressure that occur because of the normally intermittent nature of the opening of the eustachian tube. Several methods (swallowing, positive and negative Valsalva procedures, Toynbee's procedure, contraction of the tensor veli palatini muscle, etc.) were used to produce voluntarily small pressure changes in the middle ear. The differences in the hearing thresholds before and after these voluntary changes were similar to those found in the first two groups of control experiments and confirm the idea that these also were caused by the intermittent opening of the tube. Further confirmation of the idea is afforded by the fact that these procedures did not cause any changes in hearing in patients with abnormally patent tubes or in patients with severe occlusion of the tube. In Fig. 6 are shown the observations in an experiment in which the permeability of one eustachian tube of the author was increased by application of ephedrine to its pharyngeal orifice.

DISCUSSION

Opening of the eustachian tube, with resultant ventilation of the middle ear, normally occurs only during the act of swallowing. However, even when the tube is normal, complete equalization of middle-ear with atmospheric pressure is probably of short duration, because resorption of the air in the middle ear is a continuous process. The degree of negative pressure developed during any given time interval after swallowing differs in individuals, being influenced, among other things, by the factors of differences in volume of air, in surface area of the mucosa of the middle ear plus mastoid cells, and of vascularity of the mucosa. Other variables that affect the pressure at any time are conditions of the nasopharynx or the tube which interfere with equalization of middle-ear and atmospheric pressures during swallowing. In patients with such lesions, either acute or chronic, secondary changes in the middle ear introduce yet more factors. The evaluation of the effect on hearing of the separate factors is in patients often impossible because of the large number of variables. This fact probably accounts for the differences in the beliefs of otologists with regard to the effects of tubal occlusion on hearing.

In the occlusion experiments reported in this paper success seems to have been attained in the attempt to limit the number of variables to one; namely, pressure in the middle ear. Although it was not possible to avoid making occasional swallowing movements, the presence of the inflated balloon in the nasopharynx effectively prevented equalization of pressures, so that the pressure in the middle

ear fell progressively throughout the experiment. If significant secondary changes in the middle ear had occurred, it is most improbable that the hearing would have returned to its original levels for all frequencies promptly after restoration of normal ventilation of the middle ear.

Clinically, these experiments serve to emphasize the importance of early recognition and treatment of tubal occlusion. A patient with impaired hearing for high tones and tubal occlusion should be given the benefit of any doubt as to the cause of the impairment—occlusion, inner-ear lesion, etc.—and prompt measures should be instituted to restore normal ventilation of the middle ear.

SUMMARY AND CONCLUSIONS

The effect of simple occlusion of the eustachian tube on hearing was investigated. Inflation of a soft rubber balloon in the nasopharynx was used to obstruct the tubes. The hearing was repeatedly tested during the next one and one-half hours, using a Western Electric Co. 1-A audiometer that produces frequencies from 32 to 16,384 cycles per second.

This purely mechanical occlusion of the eustachian tube caused first and mostly a hearing impairment in the high-tone region. Later the hearing for middle and low tones became slightly impaired. Immediately after restoration of adequate ventilation of the middle ear (removal of the balloon, positive Valsalva procedure and swallowing) the hearing threshold for all frequencies returned to the values obtained before placement of the balloon.

The prompt recovery of hearing acuity proves that secondary changes, such as edema of the organ of Corti or of the round window membrane, could not have been responsible for the impairments, which can only be explained as a result of pressure changes in the middle ear following the experimental occlusion.

These experiments confirm the clinical impression of Crowe and Guild that an uncomplicated tubal occlusion causes a hearing loss for high tones, sometimes followed by impairments for other tones also.

Tubal occlusion must be considered as one of the causes of impaired hearing for high tones. The fact that the original hearing can be restored emphasizes the importance of early recognition and treatment of patients with this cause of impaired hearing.

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XXXVIII

CHRONIC GRANULAR PHARYNGITIS

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NASHUA, N. H.

This study of chronic pharyngitis will be limited to that of the oropharynx. The nasopharynx and the hypopharynx vary in anatomical and histological structure from it. The former is lined with ciliated epithelium, forms the bed of the adenoids, and presents the pharyngeal opening of the eustachian tubes; the latter presents the orifices of the larynx and esophagus. These two are subject to diseases that differ in character from those affecting the midpharynx proper. Here the mucous membrane consists of squamous epithelium and numerous mucous glands and lymph nodes which may become hypertrophied and infected. This membrane is highly vascularized and enervated. It has the double duty of transmitting air and food and is subjected to infections and trauma by way of the mouth as well as from the nose and nasopharynx.

Diseases of the tonsils, neoplasms, acute infections of the pharynx, tuberculosis, and syphilitic pharyngitis are eliminated from this study.

PATHOLOGY

Chronic granular pharyngitis is characterized by an alteration in the mucous membrane of the pharynx involving the mucous glands and the lymph follicles. The initial stage of hyperemia is followed by an inflammatory change in the connective tissue and the hyperplasia of the lymphoid follicles. The granulations are largely made up of masses of lymphoid cells, grouped around the mouths of the ducts of the mucous glands; they vary in size and number in different persons. The epithelium becomes thickened in some places and atrophied in others.

Schenck[1] states that the histologic section of tissue from the pharynges of over 100 patients presenting chronic pharyngitis invariably presented evidence of hyperplasia of the lymph nodules and chronic inflammation. These histopathologic changes were common to all types of chronic pharyngitis regardless of the variations in systemic complications.

ETIOLOGY

In chronic pharyngitis search should be diligently made for some local or general cause, and better results will be obtained if we are good diagnosticians than if we merely apply local remedies.[2] Among the various etiological factors of chronic granular pharyngitis, the one most commonly stated is that of postnasal discharge emanating from the accessory nasal sinuses. While this is undoubtedly a factor in a number of such cases, there are numerous other causes. LeJeune,[3] Schenck,[1] and Lillie[4] do not believe that chronic pharyngitis is always due to constant bathing of inflammatory material from the sinuses above or the lung below, and Lillie states that his "observations have led him to believe that the most frequent cause of pharyngeal complaints for which patients seek advice is referable to the pharynx itself."

There are undoubtedly many patients presenting granular pharyngitis which follows relatively closely upon an operation for the removal of tonsils or adenoids, or both. Here it appears as if nature were compensating for and replacing these areas of lymphoid tissue which the original surgery has removed. Mouth breathers, from whatever cause, usually nasal obstruction, frequently present a pharyngeal wall studded with hypertrophied lymphoid follicles which is probably due to dryness resulting from the inspired air's absorbing moisture from the pharyngeal mucosa, dilatation of capillaries, and local inflammation.[5]

Exposure to dust and irritating gases accounts for a certain number of cases of chronic pharyngitis in those connected with such occupations as stone cutting and cigarette manufacture and those in the asbestos industry.[6] Here the chronic pharyngitis is probably due to recurrent acute attacks. Among the noninfectious irritants, tobacco smoke is a common source of irritation to the pharynxes of certain individuals. This can be demonstrated by observing an irritated pharynx, accompanied by a cough, subside after smoking is eliminated. Johnson[7] states that dental caries is always a considerable factor.

Some general systemic diseases such as constipation, rheumatism, hepatic cirrhosis, and cardiac affections may predispose to chronic pharyngitis, due either to the accompanying toxicity or to disturbances in the circulation. There is a group of patients, with otherwise negative findings, whose symptoms and pharyngeal appearance suggest dietary errors, faulty metabolism, or endocrine dyscrasias. On the other hand the pharyngitis of allergy is described by Hansel[8] as

a diffuse redness and edema; rarely are there any granulations present unless complications exist.

SYMPTOMATOLOGY

The most constant symptom is that of a sore throat, especially when the patient is fatigued, a dry hacking cough, and occasional hoarseness, probably due to the thick tenacious secretions which consist of collections of mucus in clumps. Inspection reveals a diffuse injection of the pharyngeal mucosa, and nodules of lymphoid tissue distributed over the pharyngeal wall, varying in character and in number with different individuals, and with the same individuals at various times. At times these are markedly inflamed and occasionally present crypts which may contain cheesy like collections; the intervening membrane may be pale and even atrophied in appearance, or dry and glazed. The blood vessels are usually enlarged. The mucous glands and the lymph nodules are especially conspicuous behind the posterior faucial pillars, because they are more closely grouped along the lateral pharyngeal wall.

In diagnosing a granular pharyngitis a thorough inspection must be conducted carefully and extensively in order to eliminate the purely catarrhal type which may be due to sinus disease, and care must be taken that some pathologic factor in the larynx or the postnasal space or even in the pulmonary system is not overlooked.

TREATMENT

That no specific therapy has as yet been accepted for this condition is evidenced by the fact that so many varied measures are adopted for its relief.

Probably the simplest treatment is that of normal saline nasal and postnasal wash, once or twice a day. Shambaugh[9] recommends this, stating that it will relieve the symptoms. Many authorities disagree and do not find any relief of subjective symptoms nor change in the pharyngeal appearance following either douches or gargles.

Eidinow[10] has devised a quartz mercury vapor throat lamp with which he reports encouraging results with chronic inflammatory lesions of the pharynx and nasopharynx. He described the reaction as that of initial hyperemia and vasodilatation, followed by absorption of the inflamed follicles.

Hindley-Smith[11] recommends ultraviolet therapy with the cold quartz lamp, reporting gratifying results in chronic pharyngitis.

Eagle and Reeves[12] report the successful treatment of nonencapsulated lymphoid tissue of the pharynx by roentgen ray radiation: "We suggest such treatment in all patients with chronic symptoms referable to hypertrophy and infected pharyngeal lymphoid tissue, especially when other modes of treatment have failed to alleviate those symptoms."

In April, 1937, Reeves[13] reported approximately 300 patients who had received radiation therapy for chronic pharyngitis. He described the reaction as characterized by congestion and swelling of lymphatic nodules shortly after the treatment. Later the pharyngeal follicles lose their reddened granular appearance and are replaced by fibrous tissue. He advises fractional doses of low voltage, in the same manner as when treating chronic infections elsewhere in the body.

Richards[14] states that in several instances he has found multiple small doses of X-ray effective in this condition.

Gordon[15] advises that vaccine therapy be employed to clear up the local pharyngitis and to deal with the systemic complications when present.

Electrocoagulation to the individual follicles has been recommended by Schenck[16] and others.

Schenck,[16] Carmack,[17] Lillie,[4] and various others favor local caustics, such as silver nitrate and chromic acid. Some surgeons recommend surgical removal under local anesthesia. All of these therapeutic measures are variably successful. Nearly all authorities recommend internal medication with iodine in some form or other, either as Lugol's solution, potassium iodide, lipoiodine, or even syrup of hydriodic acid. Lillie[4] reports satisfactory results with the ingestion of iodine in a tablet form given two to four times a day, the duration of medication being governed by the amount of relief experienced by the patient.

During the past two years I have made a study of 29 patients with symptoms of granular pharyngitis, all of whom presented the typical nodules of hypertrophied lymphoid tissue distributed in varying amounts over the pharyngeal mucosa. All but one of these patients had previously undergone tonsillectomy. The youngest patient was 11 years of age, the oldest was 67. Most of the patients were between 15 and 40 years of age.

The treatment consisted of prescribing an aqueous solution of iodine internally, and either cauterization with 50 per cent silver

nitrate solution or destruction by means of electrocoagulation. This local treatment was applied to the larger nodules, care being taken not to destroy surrounding or intervening mucous membrane.

Iodine has been generally recommended and I have secured almost uniformly good results with it. Since it is well known that iodine lowers the basal metabolic rate in hyperthyroidism, these patients were all subjected to a basal metabolism test before therapeutic measures were instituted. I was seeking to determine the possibility of a disturbance in iodine or thyroid metabolism as an etiological or concurrent factor in this condition. (See Table I.)

Twenty-six of these patients studied had a basal metabolism rate of between +7 and −10 per cent. One patient with a rate of +11 was a middle-aged female with mild symptoms of thyrotoxicosis, her rate being reduced to −11 per cent while undergoing treatment. Two young adults with rates of −16 per cent also complained of symptoms of hypogonadism; these two patients received small doses of iodine and were referred to an internist for further endocrine therapy following an improvement of their throat symptoms. One young woman, 18 years of age, with an especially large amount of pharyngeal lymphoid tissue which did not respond to the above treatment is now undergoing X-ray radiation. As the treatment is not completed, results cannot yet be ascertained.

Although this series is too small to be conclusive I can find no evidence that disturbed iodine metabolism plays any part with granular pharyngitis, as a basal metabolic rate between +10 and −10 is considered normal. In order to supplement this study I spent a few days in a well-known endocrine clinic where I was informed that the patients, either with hyperthyroidism or hypothyroidism, did not complain of pharyngeal symptoms as such. I had the occasion to examine several of the patients and found no more pharyngeal pathology than one would expect in any similar group in any office or clinic.

The favorable results from iodine therapy are probably empirical, possibly biochemical, and do not serve to readjust a disturbed iodine metabolism.

SUMMARY

1. The basal metabolic rate of patients with granular pharyngitis in a series of 29 cases was within normal range in all but 3 cases, and these presented complicating endocrine disturbances.

2. Patients with hyperthyroidism or hypothyroidism, as a group, did not complain of pharyngeal symptoms more than did any control group.

3. Symptoms of granular pharyngitis are improved by local treatment and internal iodine therapy.

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TABLE I

SUMMARY OF BASAL METABOLIC RATES

Initial	Sex	Age	Symptoms	B.M.R.	Appearance of Pharynx, Amount of Lymphoid Tissue	
A. P.	F.	67	Cough, fullness in throat, dysphagia	—4	++	
A. G.	M.	23	Dysphagia and dry cough	+2	++	
J. C.	F.	19	Tightness in throat and cough	—4	++	
M. F.	F.	17	Dysphagia, fullness in throat	—9	+++	
E. W.	F.	51	Frequent sore throats	—7	++	
A. G.	F.	19	Sore throat and cough	—2	++	
P. W.	M.	22	Cough and dysphagia	+8	+++	
K. G.	F.	34	Dry cough and dysphagia	—7	++	Tonsils present
B. M.	F.	44	Cough, dysphagia	+11 and —11	++	
A. B.	F.	36	Constant sore throat and cough	+5	+++	
E. B.	F.	21	Recurrent sore throats	—1	+++	
R. S.	M.	13	Cough and sore throat	+4	++	
E. H.	F.	38	Dysphagia and dry cough	—6	++	
B. D.	F.	16(?)	Dry cough and sore throat	—10	+++	Undergoing X-ray treatment
M. L.	F.	17	Frequent sore throats and colds	—3	+++	

TABLE I (CONTINUED)

Initial	Sex	Age	Symptoms	B.M.R.	Appearance of Pharynx, Amount of Lymphoid Tissue
C. P.	M.	14	Sore throat and frequent colds	—4	++
E. S.	M.	43	Cough and dysphagia	—2	++
H. W.	M.	38	Dry cough, fullness in throat	+3	++
J. B.	F.	18	Cough and sore throat	—6	++
M. J.	F.	22	Cough and sore throat	—5	+++
R. H.	M.	25	Dry cough and sore throat	—4	++
S. M.	M.	13	Frequent colds and sore throat	+8	++
L. G.	F.	22	Dry cough and dysphagia (hypogonadism)	—16	++
A. J.	F.	31	Colds and dry cough	+2	++
K. B.	F.	28	Dry cough and sore throat	+3	++
E. W.	M.	37	Fullness in throat and cough	+1	+++
R. K.	M.	31	Dysphagia and cough	—4	++
R. P.	M.	43	Frequent colds and sore throat	—1	++
C. P.	M.	18	Cough and sore throat (hypogonadism)	—16	++

XXXIX

FRACTURES OF THE FACE INVOLVING NASAL ACCESSORY SINUSES*

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During the present national emergency many maxillofacial, oral, and plastic surgeons will be called into service and it seems an opportune time to bring before this section a discussion of fractures of the facial bones, inasmuch as this may be considered borderline surgery. Maxillofacial surgery had its greatest development during the last war, and after the war because of the great increase in automobile production, speed, and consequent accidents, maxillofacial surgeons found a permanent and increasing field of work. It is reasonable to presume that in the smaller communities, and perhaps in the larger centers, competent maxillofacial surgeons will not be available in the near future. Inasmuch as the nasal accessory sinuses, the orbit, and the nose are involved as a rule in fractures of the face we believe the rhinologist should familiarize himself with the pathology, complications, and treatment of these fractures. It must always be kept in mind that the cosmetic as well as the surgical results will be of tremendous importance to the patients after their recovery.

In 1918 we noted in a paper on "Fractures of the Face" that only a small proportion were due to automobile accidents, but in 1928 in a paper presented before the middle section of the Triological the majority of cases seen were due to automobile accidents. This discussion will consider the type of injury sustained in automobile and industrial accidents, i.e., crushing injuries of the facial bones with or without lacerations of the soft tissues. These fractures differ from war injuries in that the latter are caused as a rule by flying missiles.

The majority of automobile accidents are seen by general surgeons at emergency hospitals and it is reasonable to suppose that the more obvious injuries and fractures and the general condition

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*Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Feb. 1, 1942, Los Angeles, Calif.

of the patient will receive first attention. Edema, hematoma, and emphysema, all of which may occur early, may obscure facial bone fractures.

In 1928 inasmuch as we were rather at a loss to know just what structures were involved in fractures of the face and also to give us suggestions as to the methods to be used in restoring function and normal appearance in these cases, we made some experiments on the cadaver, trying to reproduce as nearly as possible the usual automobile fractures. The following are a few of the experiments.

1. Force was applied directly to the lateral surface of the malar, resulting in dislocation of the malar from the frontal and zygoma. There was a fracture of the facial surface of the maxillary sinus and the orbital floor, and the orbital process of the malar was driven into the maxillary sinus.

2. The blow was applied to the infraorbital ridge with the following result: the frontal process of the malar was broken below the articulation with the frontal; the facial surface of the maxilla was fractured vertically; the zygoma was fractured at its junction with the squamous portion of the temporal and there was a lateral displacement of the zygomatic arch.

3. Force was applied to the bridge of the nose, with the result that the nose was crushed with a buckling upward of the nasal septum causing a fracture of the cribriform plate and loosening of the cristi galli; there was a separation of the frontal process of the maxilla from the nasal process of the frontal, thus opening the floor of the frontal sinus. There was a comminuted fracture of the ethmoid labyrinth and the pterygoid process was broken from the body of the sphenoid, thus opening the sphenoid sinus.

4. Force was applied to the alveolar process from in front similar to the injury sustained when the victim is thrown against a dash or back of the front seat of an automobile. As a result both maxilla were fractured horizontally through their bodies, thus exposing the maxillary sinuses. The nasal septum was fractured from before, backward through the septal cartilage and vomer.

Following these experiments the heads were dissected and lines of the fractures studied. Many inaccuracies can be found in these experiments: (1) No definite measurements of the force applied or its direction can be made, and there would be a variation in the

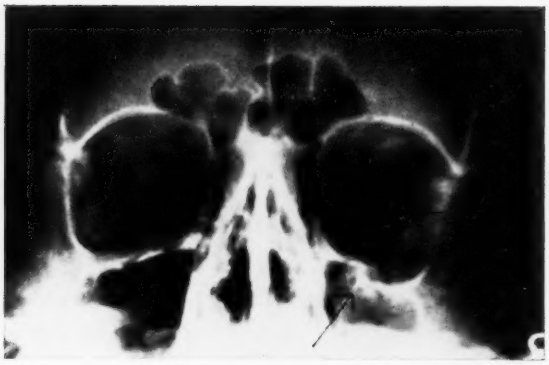

Fig. 1.—Fracture of orbit showing narrowing and depression of infraorbital ridge.

type and shape of the implement used in comparison with accidents. (2) The age of the patient has to be considered, as it influences the density of the bone, the aeration of the sinuses, and the ossification of the suture lines. No claim to priority is made for this research as we noted recently where La Forte, a French surgeon, made similar experiments in 1901. However a study of these experimental fractures led to a more rational method of treatment and explained some of the unpleasant complications which sometimes occur. Important among these observations was that the facial wall of the maxilla and the orbital floor are almost invariably fractured in blows on the malar eminence. Second, that in crushing blows on the nose the cribriform plate may be fractured with displacement of the cristi galli. In blows on the alveolus from the front, the floor of the maxillary sinus is opened. It was also observed that in comminuted fractures of the wall of the maxillary sinus no effort is needed to enter the antrum, simply incise the mucosa above the alveolus and the antrum can be entered through the comminuted bone without the use of the gouge or drill.

Fractures of orbit and orbital ridge can be diagnosed by palpation, crepitus, and the X-ray and there may also be a displacement of the eye downward. In fractures of the malar, as pointed out by Tholen, the zygoma can be palpated by placing one finger in the mouth and another over the zygoma and as a rule crepitus

can be elicited. It should be kept in mind that edema, hematoma, and emphysema will alter the contour of the normal anatomy as well as obscure fractures. Complete roentgenograms should be taken in all positions, particularly including the malar vertex position, and these roentgenograms should be repeated following the reduction of the fractures. Roentgenograms are of particular value in malar fractures and fractures of the orbit. In the latter there is usually a narrowing of the horizontal axis of the orbit and an overlapping of the ridge. Roentgenograms of the nose should always be taken and should be rechecked later as they may give considerable information. It is true they often seem of little value as far as treatment is concerned. A roentgenogram is also of great value in transverse fractures of the superior maxilla. In fractures of the wall of the maxillary sinus there is usually a cloudy sinus because of the blood in the antrum.

In regard to treatment the final cosmetic results must always be kept in mind as well as the normal physiologic and functional results. This may mean much to the patient both from a social and economic standpoint. Do not forget that your heroic life-saving efforts may be entirely overshadowed later by the contemplation of an unsightly cosmetic end result, that is scars and facial deformities. With that in mind immediate careful repair of the soft tissue lacerations is imperative. Thorough cleansing of the wound, excision of badly lacerated tissues, subcutaneous or fine silk sutures are advised and the sutures should be removed in three or four days. The lacerated wound should be examined carefully for foreign bodies, particularly glass, as on several occasions I have found particles of glass which were overlooked and sutured in the wound during emergency surgery. All hemorrhage must be controlled before suturing. Pressure dressings will help control bleeding but obviously little pressure can be used over a badly comminuted anterior wall of the maxillary sinus or nose. If the condition of the patient permits, immediate reduction of the fracture is advised. Early reduction and immobilization of the fractured parts reduces shock, relieves pain, controls hemorrhage, gives a more rapid restoration of function, and prevents deformities.

Where shock or great loss of blood or other more serious injuries exist it is best to postpone reduction of the fractured facial bones until the condition of the patient improves. Always keep in mind however that because of the abundant blood supply of the face, union takes place early and in ten days to two weeks quite firm union exists. Therefore the operation should not be postponed beyond this period.

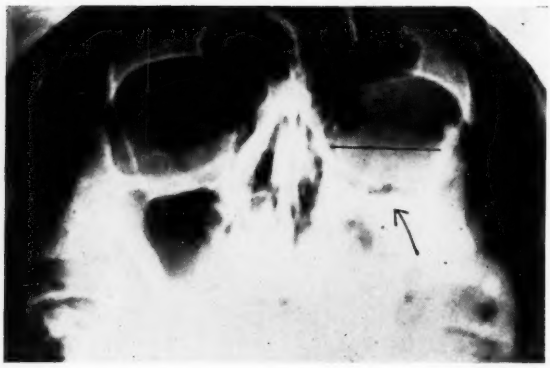

Fig. 2.—Fracture of orbit showing narrowing of orbit.

If the alveolar process or the teeth are involved and there is a transverse fracture of the maxilla with separation of the skull, a dental or oral surgeon should take charge as he is more familiar with the dental splints and appliances required. When teeth are in place interdental wiring can be used and in edentulous patients, plates can be used. For emergencies the use of the four-tailed Gibson bandage is of value to immobilize the parts.

The plaster head cast is very essential in extensive transverse fractures of the face to provide supports which are either wired to the teeth or attached to splints placed in the mouth. These same supports can be used in fractures of the zygoma and in comminuted fractures of the nose and also where continuous pressure is to be exerted against the side of the nose or malar bone. Stout rubber bands may be attached to the wire supports in order to exert continuous traction. As pointed out by New, continuous traction is often needed to pull fractures in place and get proper occlusion. These head casts, as pointed out by Ivory,[2] may be made by pulling a stockinette over the head. This is brought down below the occipital protuberance and just above the ears and eyebrows. The upper end is tied on top of the head; four strips of othopedic felt, six inches long and one inch wide, are placed vertically over the head, one in each quadrant, and held in place with adhesive strips. A three-inch plaster bandage is then applied as a head bandage and hooks cut from coat hangers or wire are incorporated in the plaster

to be used as traction posts for rubber bands which are attached to the splints. These supports may be placed in the nostril in comminuted fractures of the nose and in the mouth where there is a transverse fracture of the maxilla. Fractured nasal bones are as a rule easy to replace but in badly comminuted fractures may not stay in place, and it is sometimes necessary to use considerable force with a blunt instrument from within the nose to replace a fractured nasal process. A large septum forceps, such as an Ash's septum-straightening forceps, is of great value in replacing a dislocated fractured nose. It has been advised by New[1] and others, in badly comminuted fractures of the nose, to delay operation for ten days because of the possibility of meningitis. However if the patient can be seen immediately, that is within a few hours following the injury and if the bleeding can be controlled, we believe that replacing the fractured bones and thereby obtaining better drainage is good surgery. If the patient is not seen until several days following the accident it would be well to delay proceedings until there was no evidence of infection.

Fractured nasal bones can be kept in place as a rule by intranasal and extranasal splints. In badly comminuted cases where the septum and nasal processes are fractured and the nose flattened it is best to attach wire splints to the plaster head cast. Intranasally the wire supports are covered with dental compound so as not to injure the nasal mucosa. A wire suture may be put through the nose transversely and the ends of this wire are then attached to the head cast. In my experience these wires have had a tendency to cut into the skin and infection may develop.

The malar bone forms part of the lateral wall and floor of the orbit and articulates with the superior maxillary and with the zygomatic process of the temporal. In impacted fractures where the force is applied to the malar eminence, the orbit and maxillary sinus are as a rule fractured. The ethmoid and nasal bones may sustain fractures also.

The treatment of fractures of the malar varies with the type of fracture. In simple fractures and displacement of the body of the malar where the bone is not comminuted, the malar may often be elevated by grasping the bone through the skin with a stout tenaculum forceps and gradually rotating the bone into place, or a small incision may be made in the skin and the bone elevated with a sharp hook. When the zygoma is fractured an incision may be made through the skin and muscle in front of the top of the ear;

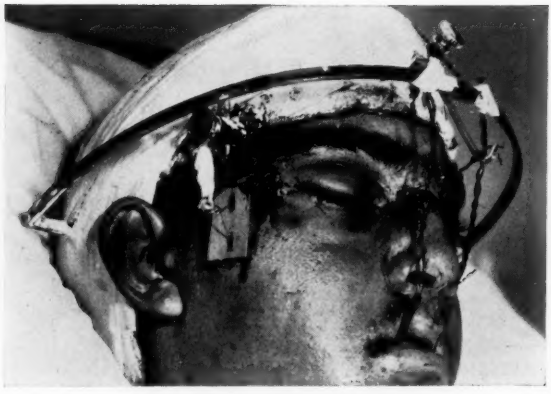

Fig. 3.—Head cast with attachments. (Courtesy of Dr. E. Tholen.)

then the hemostat is pushed down until it is beneath the zygoma; a roll of gauze is placed between the instrument and the head and the bone brought into place. Where the arch is fractured and the malar depressed a method suggested by Corbin[3] may be of value. The cheek is drawn back and an incision made through the mucosa and submucosa in the buccal sulcus above the molars. A hemostat is used to create a tunnel to the fractured bone; a thick blunt instrument is introduced into this tract until it touches the malar bone. The position of the instrument is then medial and inferior to the zygomatic arch. With the handle firmly grasped and using the skull as a fulcrum, the bone can be elevated. This can also be accomplished in some cases by putting a finger in the mouth under the cheek and pushing up on the zygoma.

In our experience in the majority of fractures of the malar where the anterior wall of the maxillary sinus is comminuted, there is also a fracture of the outer wall of the orbit and infraorbital ridge. In this type we make an incision through the mucosa under the lip as in an antrum operation and usually find it possible to enter the antrum by removing particles of comminuted bone. If this is not possible an opening is made with the gouge. I prefer a vertical incision through the mucous membrane in the region of the bicuspids in any type of external antrum operation as this avoids

injury to the nerve supply of the face and the resulting numbness which often occurs. We have found in making this vertical incision for an antrum operation that the patient seldom complains of numbness in the lip and cheek after the effects of the local anesthesia have disappeared. After the antrum is opened a stout blunt instrument, such as a curved Kelly sound, is introduced and the malar is pushed up into place, using care not to displace particles of bone or force the instrument through the antrum wall. In some cases it is possible to grasp the bone through the skin with a stout tenaculum to assist in the manipulation and it is often possible to assist in this by putting the finger into the mouth and pushing up on the zygoma. Fragments of the comminuted antrum wall are carefully protected unless they are practically torn loose from the mucosa. As a rule these fragments will unite if pushed up into place as the blood supply of the face is abundant and necrosis of the fractured bone seldom occurs. As a rule we do not advise any packing in fractures of the malar or fractures involving the maxillary antrum and malar unless the anterior wall of the antrum is so badly comminuted that the fragments of the bone will not stay in place. We have found that in the majority of cases if the fracture is properly reduced the bones will stay in place. When packing is needed because of a badly comminuted anterior wall of the antrum or floor of the orbit with displacement of the eye downward which will not stay in place, we prefer vaseline gauze packing which may be covered with a rubber dam. The packing is then left in the antrum for from four to six days and as a rule it is not necessary to repack it. In extreme cases a wire splint can be introduced into the opening into the antrum to push up against the floor of the orbit if the orbital floor cannot be kept in position by packing. This necessitates very careful adjustment of the splint and attachment to the plaster head cast. At times wiring may be necessary to hold the fractured ends of the malar or orbital bones in place. This requires an incision through the skin over the bone and will result in scarring. In order to provide drainage for the infection which usually develops in the antrum it has been our custom to make an antrotomy opening under the inferior turbinate. This will permit immediate closing of the wound under the cheek if there is no packing, and in the event that packing has been used the wound can be closed as soon as the packing is removed and subsequent treatment can be carried on through the nose. We do not believe in frequent irrigations of the antrum following these operations. One irrigation a few days following the operation is advisable to remove clots. One of the sulfanilamides in powder

form may be used in the antrum or in the wound where there is a great possibility of infection. It should be given internally where there is a suspicion of meningitis or systemic infection. A prophylactic dose of tetanus antitoxin is given in all cases with lacerations.

In fractures which involve the ethmoid we may have an emphysema of the orbit due to fracture of the orbital plate. Treatment of fractures of the ethmoid are directed toward drainage, therefore reduction of fractures of the nasal bones and installation of neosynephrin or some similar drug is indicated. In fractures involving the frontal sinus we believe in conservatism unless there is a compound fracture of the anterior wall. In compound fractures of the frontal sinus the wound should be cleaned thoroughly and drainage put into the frontal sinus. In simple fractures of the frontal, even if the X-ray showed some blood in the frontal, we have followed conservative treatment unless symptoms of infection developed. By encouraging intranasal drainage by the use of shrinking solutions, drainage may be established from the frontal, but should symptoms of infection develop an external operation is the only one indicated in these cases, as in the external operation inspection can be made of the posterior wall of the frontal sinus.

Because of injuries to the peripheral nerves, manipulation of the fractured bones of the face can usually be carried out with little pain, and this is an added advantage of early reduction. Local anesthesia with nerve blocking will suffice in nearly all of these facial fractures.

Numbness of the cheek and lip may be present for several weeks following the injuries because of injury to the infraorbital nerve and this may later be followed by a neuritis. Purulent dacrocystitis may develop from injury to the lacrimal ducts. Diplopia may exist following an injury; this is due to a displacement of the eyeball from a fracture of the floor of the orbit or to an injury to the eye muscles. Malocclusion of the teeth may occur; particles of bone may be extruded at times from a comminuted maxillary sinus and New calls attention to the fact that an osteomyelitis may develop.

SUMMARY

A careful survey of the fracture should be made at the first examination and complete roentgenograms should be obtained before and after the operation. Bleeding from the ear or nose or in the orbit may indicate a skull fracture.

If the patient's condition permits, an immediate or early suturing of the laceration and reduction of the fractures are advised. All hemorrhage should be controlled before suturing lacerations and all bone possible saved in comminuted fractures of the orbital floor and maxillary sinus. Packing in the maxillary sinus should not be used if it can be avoided.

The patients should have the benefit of plastic surgery if unsightly scars and deformities exist following their recovery.

6777 Hollywood Blvd.

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XL

GUNSHOT WOUNDS OF FRONTAL AND TEMPORAL BONES*

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CHICAGO

The following is a report of two cases of gunshot wounds which were treated in the course of my duties as otolaryngologist to the Illinois State Penitentiaries.

The first case is that of a gunshot wound of the frontal bone. The patient, A. B., male, aged 42 years, had inflicted a gunshot wound on himself on Aug. 16, 1940. From May to August, 1940, he had been a patient at the Chicago Municipal Tuberculosis Sanitarium. Shortly after his release he shot himself. The wound did not cause him to lose consciousness and he was taken to Bridewell hospital. He was first seen by me on April 24, 1941, his complaint at that time being persistent headache in the frontal area. Examination revealed a scar in the right temple, which the patient attributed to the entrance of a bullet. A firm swelling, about 2 cm. in diameter, was present in the midline of the forehead at the level of the hairline. The nose was normal on examination. The ear and throat examination was essentially negative. There were no chest signs at this time.

X-ray revealed a large dum-dum spread type of bullet embedded in the frontal bone slightly to the left of the midline, surrounded by numerous fragments. The sinuses did not appear to be involved, but there was an apparent fracture line involving a good portion of the frontal bone with bone absorptive changes present in the portion penetrated. One leaf of the bullet apparently had penetrated the inner table of the frontal bone. Because of the persistent headache and bone absorptive changes seen by X-ray, surgery was advised.

On May 1, 1941, under local anesthesia supplemented by morphine, amytal, and scopolamine, a three-inch incision was performed at the level of the hairline down through the gala aponeurotica.

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*Presented before the Chicago Laryngological and Otological Society, Nov. 3, 1941.

On exposing the bone, a large lead bullet was found, surrounded by a soft necrotic type of bone. A leaf of this bullet had penetrated the inner table and dural attachments. The surrounding necrotic bone, down to the frontal sinus and about the bullet for about 1½ inches in each direction, was removed. The frontal sinus was not entered. The bullet could then be extracted. The leaf which penetrated the dura was found lying in the frontal lobe of the brain. There were a few cubic centimeters of secretion within the encapsulated space about the bullet in the frontal lobe. Apparently a small frontal lobe abscess had formed about the projectile and had sealed off from the surrounding arachnoid space with fibrous tissue about the inserting leaf of the bullet. There was no injury to the capsule performed in removing the bullet, and essentially the removal of the bullet accomplished a marsupialization of the brain abscess. This was packed with vaseline gauze and the incision left open. The patient left the operating room in good condition. He was placed on sulfathiazole, 15 gr., every four hours. His highest postoperative temperature was 101°, and he was temperature-free on the fifth day. Sulfathiazole was discontinued on the eighth day. No skin sutures had been applied, a secondary closure being intended when the wound was well granulated. However, in about six weeks the wound had completely healed with granulations, in spite of repeated insertion of drains, and the epithelium had healed with such a narrow scar that secondary closure never became necessary. The patient has remained symptom-free to date.

During a previous imprisonment prior to the gunshot wound the patient's intelligence was rated as dull, and he was a chronic alcoholic; after the hospitalization with the bullet wound his intelligence and personality tests showed no change.

The second case is that of gunshot wound of the temporal bone. The patient, J. D., male, aged 38 years, stated that he was well and healthy until July 27, 1940. On that date he suffered gunshot wounds in the chest, left elbow, and left temple. He stated that he was rendered unconscious immediately (at 9:45 A. M.) from his injuries, and did not recover consciousness until the afternoon of the same day, when he was in a hospital. There was hemorrhage from the left ear, accompanied by a constant buzzing and dizziness. Since the date of the accident, the patient has suffered total deafness, a persistent discharge from the ear with occasional bleeding, a complete facial paralysis, and constant headaches and dizziness. The hearing test showed complete absence of air and bone conduction in the left ear, and normal hearing in the right ear.

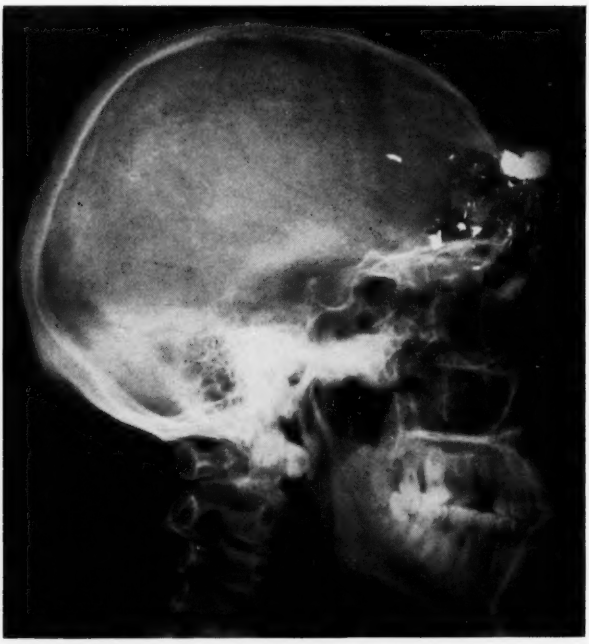

Fig. 1. Case 1.—Roentgenogram right lateral view of the skull. Bullet fragments in the frontal bone.

The fistula test of the left ear was negative. Ether in the left ear gave no reaction, but when it was instilled in the right ear nystagmus started in 20 seconds and lasted for 55 seconds.

The blood Wassermann was negative. The red blood count was 4,800,000, the white blood count 35,000, and hemoglobin 65 per cent.

The right ear drum was normal. The left membrana tympani was absent and the middle ear was obliterated by a large bullet.

The nose and throat were normal.

X-rays showed that the right mastoid was normal and of pneumatic type. The left mastoid revealed the loss of bone structure in the area above the middle ear, including the posterior part of the zygoma and squamous portion of the temporal bone about one inch in diameter. There was an absorption of trabecular struc-

ture in the mastoid. The middle ear, cochlea, and adjacent portions of the vestibular apparatus were obliterated by a radio-opaque mass surrounded by particulate radio-opaque fragments.

On Feb. 6, 1941, under ether anesthesia, Lempert endaural radical incisions were performed, exposing a normal mastoid cortex. Lateral attic necrosis was observed on exposing the posterior superior canal wall. The mastoid cortex was removed by burring out the superior and posterior bony canal wall and continuing outward over the mastoid process. No evidences of gunshot particulate matter were found in the mastoid proper, but the previously normal pneumatic bone was seen to have undergone a low-grade osteitis. The cell structures were filled with serum. Vestiges of trabecular structure remained which, when curetted, broke away in such softness as to give the impression that the bone structure had been completely decalcified. There was a complete absence of granulations. A partial mastoidectomy was completed, leaving uninvolved tip and retrosinus cells. The antrum was found filled by a large lead bullet as well as several smaller particles. In order to dislodge this major portion of the bullet, the dural plate was removed, going forward into the epitympanum. In this area a dime-sized defect was found in the dural plate and the posterior part of the zygoma. The dura in this area was prolapsed over the bullet and covered with a necrotic type of granulation tissue. A flat ribbon elevator was used to retract the prolapsed dura, and the dural plate anterior to this was removed. The bullet and most of the fragments were then dislodged with ease. The facial nerve was seen lying free over necrotic bone in its horizontal portion. It was reddened and slightly thickened. The facial canal was removed anteriorly to the geniculate ganglion and posteriorly about halfway down its vertical portion without injury to the nerve. The eroded and necrotic portions of the promontory of the cochlea, epitympanic dome of the vestibule, and ampullar parts of the superior and lateral semicircular were removed as if one were doing a débridement of sequestrating bone. While the hypotympanum was being skeletonized a gush of spinal fluid filled the operative wound. On inspection with suction, a split in the dura was observed in the diseased portion previously discussed. Apparently the pressure of the ribbon retractor caused this split, and from the excessive and continuous flow of clear cerebrospinal fluid the source was thought to be a cisterna.

Because of the continuous and excessive flow the wound in the dura was packed off with vaseline strips, and the patient was

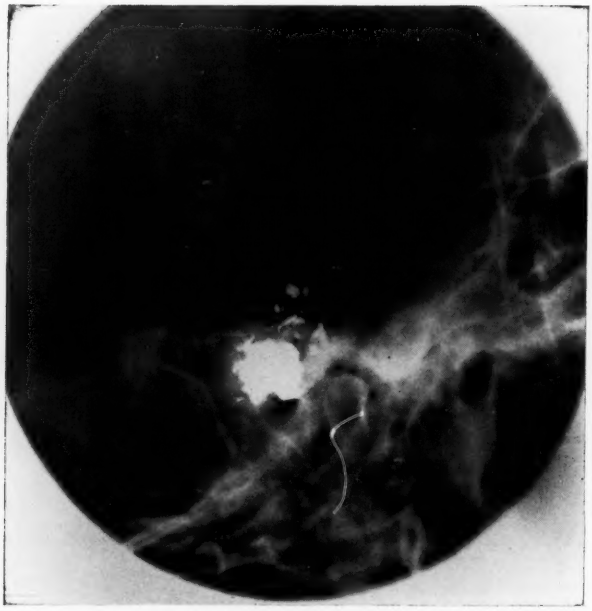

Fig. 2. Case 2.—Roentgenogram left lateral view. Bullet fragments in the temporal bone.

returned to his room in good condition. He was given 5 c.c. neoprontosil, 5 per cent, every four hours, and his postoperative course was uneventful. The temperature never went over 100.4°, and he was temperature-free on the sixth day. His red blood count on this day was 4,800,000, white blood count 8,625, and hemoglobin 70 per cent. On the eighth postoperative day the packs were removed, and apparently the dural tear had sealed off, as there was no further loss of spinal fluid. The wound healed without any mishap except for a small fistula which was practically dry and sealed off with a crust. This crust was removed from time to time and either 5 per cent sodium sulfathiazole in 70 per cent alcohol applied or sulfathiazole powder insufflated. Throughout this time the face was treated with heat and massage. About six months postoperatively the patient developed signs of returning function in his facial nerve, and to date he can close his eyelid, wrinkle his forehead incompletely, and masticate with ease. However, definite evidences of partial facial paralysis persist.

During October, 1941, the patient began to complain of neuritic pain about the ear, associated with a slight increase in discharge. External ocular muscle movements were good.

X-rays at this time revealed, besides the operative defect and the absence of the previously described bullet, the presence of mastoid cells in the unoperated tip and retrosinus cells. There were slight indications of osteal changes in the trabeculae remaining. Because of the persistent discharge and pain, reoperation was considered, particularly since the first operation had been interrupted by the dural accident.

On Oct. 30, 1941, under local anesthesia supplemented by morphine, amytal, and scopolamine, the mobile extracartilaginous endaural incisions were re-performed. The mastoid was exposed and the tip and retrosinus cells were cleaned out. Pathology here was similar to the previous pathology found in the mastoid; namely, a low-grade osteitis with serum in the cell cavities. The dural elevation in the epitympanum was found to be sealed off and was left undisturbed. Because of the apparent association of the discharge with the remaining mastoid pathology, and because of the exposed facial nerve, the only middle-ear work done was to search for cochlear and epitympanic leads to the petrous portion of the temporal bone. These were not found. The wound was packed with vaseline gauze, and the patient left the operating room in good condition.

The patient's postoperative course thus far has been uneventful. The facial nerve function is the same as prior to the second operation, and the radical ear cavity is epithelized.

310 S. Michigan Ave.

XLI

MULTIPLE BENIGN SARCOID OF THE UPPER RESPIRATORY TRACT*

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After Boeck[1] made public his memorable studies of multiple benign sarcoid, a rich literature accumulated on this subject. In the main, the notes deal with an affection of the skin. It is, indeed, the dermatologists who have contributed to most of our present-day knowledge. Other disturbances of the body were gradually added to the literature that were not of a dermatological nature per se, but which, in response to a later sifting of classifications, were found to be only other morbid manifestation of Boeck's disease. A reasonably assiduous search of the treatises, however, has failed to show any completed treatment of the upper respiratory tract. In view of the more recent studies which show that hardly any organ of the body escapes attack, at one time or another, as a consequence of this morbid process, it seems unlikely that the upper respiratory tract system would have always escaped. Accordingly, it is, perhaps, justifiable to attribute the paucity of the accounts to a lack of sufficient acquaintance with the disease. In the rhinolaryngological periodicals there are papers assaying to cover no end of tumors of the upper respiratory tract, yet a fairly diligent search fails to reveal any on the subject matter of this paper. From this point of view alone the case about to be described would be worth noting, but in addition, it appears to possess certain unusual features which are far from having had the study which they merit.

Seiferth[2] published a small tract recently entitled "Benign Miliary Lupoid Type Boeck of the Upper Respiratory Tract". A check of his paper, however, reveals only a very short clinical description of what was left of the epiglottis in his patient and a word to the effect that he also found a small elevation or two in the nose. The cicatricial mass, which purported to be the remains of the epiglottis was held in its position by a thin stem, and was so placed that it almost completely covered the entrance into the

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larynx. The larynx was not described, nor was any of its constituent structure mentioned. No word was said about bronchoscopic examination. Nor was the diagnosis based upon his own microscopic findings. From the article it can cafely be said that the diagnosis of the disease affecting his patient was made at the skin clinic, and not from any personal investigation.

He stated that the color of the skin lesion was brownish red, and was of a butterfly shape stretching from the cheek of one side across the nose to the cheek of the other side. This gives the impression that the diagnosis of the lupoid characteristics of his patient was probably correct.

A case report of a sarcoid which showed evidence of laryngeal involvement given by Allison and Mikell[3] can be ruled as outside the sphere of this paper. It was an unquestioned Boeck's sarcoid of the skin, but the larynx was afflicted with frank tuberculous caseation. Hence, it cannot be regarded as sarcoid of the larynx but as frank tuberculosis, exactly as they had stated.

Pinner,[4] in his review of the literature on the subject of Boeck's sarcoid, mentions two reports in the foreign literature and one in the American literature. The latter was by Longcope and Pierson,[5] who reported "general edema of the epiglottis and laryngeal structures." This statement is of a general clinical nature, bearing no specific information to the laryngologist. It was made as a clinical finding to account for the hoarseness in a patient suffering from generalized sarcoidosis.

Novy[6] reported a case of a colored patient who suffered from frequent nosebleeds which were attributed to sarcoid infiltrations present in the nose at that time. Fletcher[7] reported a patient who had a chronic nonhealing lesion of the nose, which later studies revealed to be sarcoid.

It is partly because of the great variety of clinical forms in which this disease appears, and partly because of the interest Boeck, Besnier[8] and later Schaumann,[9] Heerfordt[10] and Jungling[11] have aroused among the medical profession that the contributions are practically innumerable and the literature has reached enormous proportions.

So far as I have been able to ascertain, both by speaking with rhinolaryngologists of wide experience, and by searching diligently the various publications that carry a description of this disease, this

paper bids fair to be the first investigation of a verified study of multiple benign sarcoid of Boeck of the upper respiratory tract.

While going through the literature on this subject, I found the following rather illuminating statement made by Dr. R. Fletcher in his paper on a case of a chronic nonhealing lesions of the nose: "I have never heard of the term sarcoid before hearing Dr. Kistner's paper in which he reported a case of sarcoid of the nose". This poignant statement indicates the correctness of the inference made at the outset that it is justifiable to attribute the paucity of the essays dealing with the upper respiratory passages in relation to this disease to lack of acquaintance with it. At no time while under Dr. Fletcher's care did the patient, an adult white female, complain of or show any evidence of cutaneous lesions. Not until the nasal lesions healed did discolored infiltrations in the skin begin to appear.

While I am mindful of the danger when one attempts to draw inferences from a study of insufficient instances, the interesting data offered by Fletcher coupled with Novy's and my own (though the latter two are more open to question) of lesions in the nose antedating those of the skin cannot be passed off without further comment, even though speculative. The lesions alluded to open up a question as to whether the upper respiratory passage (nose to bronchi) may not be the seat of the first infection.

Sarcoidosis can no longer be considered a rarity to be seen only in metropolitan clinics ministering to vast populations. Some of the patients studied came from small suburban communities to the larger clinics in their attempt to seek relief after the local professional skill was exhausted.

It may be assumed that incidence of this disease, attacking the organs which go to form the upper respiratory tract, is certainly greater than the scarcity of reports in the medical literature would indicate. Because of the usually meager personal experience available to the rhinolaryngologist, he is of necessity dependent upon the literature for guidance in the recognition of this unfortunate affliction, and in response to the presumed need, this report will be given in somewhat more detail than would otherwise be the case.

The skin lesions, which so often bring the patient to the doctor, are generally round or oval, with normal surfaces and sharply defined borders. The size may run anywhere from a tiny pinhead to a large plaque. They present as a single lesion or appear in larger numbers. The lesions may be papular, nodular or large infiltrated plaques. The color may be yellowish, bluish, or brownish

red. Sometimes upon diascopic pressure minute yellowish foci may be seen, as observed in lupus vulgaris, and it is said that it was this characteristic that led Boeck later to suggest "miliary lupoid" as a better term than sarcoid. Later in this paper I shall give an account of my experience with diascopic pressure in my patient which may offer some hints of value for further studies. Generally there are no subjective sensations of pain.

The infiltrations may be located in the face, forehead, cheeks, nose, neck, shoulders, arms, legs, abdomen, hands, fingers, etc. On palpation the plaques usually give the impression of moderately firm and elastic infiltrations in the cutis, which is freely movable over the subcutaneous tissues. In the fingers of my patient the plaques were more firmly adherent and were not as freely movable over the tissues as in other parts. In their distribution they showed an indifferent symmetry and no predilection for either flexor or extensor surfaces of the arms or legs. They sometimes tend to involute spontaneously. When that takes place they leave a brownish pigment or a loss of pigment as in my patient or an atrophic scar. The regional lymph glands are sometimes involved. Stelwagon[12] states that the malady presents in some instances a rough resemblance to lupus vulgaris and sarcoma.

In recent years a number of excellent reviews have been published and are readily accessible. These may be found in treatises on the skin, in writings on tuberculosis, or in journals for the general practitioner. None, or practically none, however, can be found in literature destined principally for the rhinolaryngologist.

Kaposi[13] was probably the first to use the term sarcoid to designate a collection of tumors of the skin. Some of the tumors which Kaposi included in his collection, which he designated "sarcoid", have been removed from the group. In 1899 Boeck published his epochal work, which has come to be known as "Multiple Benign Sarcoid of Boeck". It was a description of nontender, firm, noncaseating nodules or plaques of varied sizes in the skin. He named the disease sarcoid, because histologically some of his specimens resembled the small cells of sarcoma. Later Darier arranged a classification of the sarcoid, but this classification is of but little interest to the rhinolaryngologists, and will be omitted here. Following Boeck's publication, many clinicians added observations of their own. These latter carried descriptions of clinical manifestations differing widely with those made public by Boeck. They included descriptions of lymphogranulomatosis, cystic osteitis, disturbance of the uveal tract and parotic gland; the lacrimal gland

and salivary glands, etc. Each of these, after being made public, became known as the disease of the particular author, and at present are sometimes better known by the author's name than by the pathologic nomenclature. All were regarded as separate entities.

It is only in comparatively recent years that these clinical manifestations have been recognized as different expressions of a single pathologic process. Most workers, at present, consider tuberculosis the underlying process of this disease. Notwithstanding this almost universally accepted theory, it cannot be said that it remains unchallenged. The reason for this generally accepted version can be found in the histologic picture; indeed, show a microscopic slide to a general pathologist and he will unhesitatingly pronounce it tuberculosis. There continues, however, to be a school not in favor of the tuberculous origin of this affection. They base their argument upon the facts that the tubercle bacilli are rarely demonstrated in sections, that inoculation experiments in animals fail to produce the picture of sarcoid, and that the tuberculin reaction is usually negative. Every now and then leprosy is brought forward as playing a role in its etiology. Pinner makes a very strong argument for the tuberculous etiology in his excellent review of over 200 cases to which he added several of his own. That this disease possesses a tuberculous base has long ago been advanced. Longcope[14] recently said that in spite of innumerable contributions the cause of sarcoid, its relation to tuberculosis, its pathogenesis, its treatment and the manner by which recovery ensues remain matters of controversy and dispute.

Though the disseminated sarcoid of Boeck runs a chronic and often a lethal course, spontaneous healing of some of the lesions have been observed. In some cases the disease may last for years, while others succumb in a comparatively short time. It may go through longer or shorter periods of remissions, but it may also relapse, involving one organ after another, until eventually proving fatal.

Almost immediately after the disease was classified, the thought was advanced that the infiltrations of sarcoid were really a tuberculid. Though the tubercle bacillus avoided detection in most of the cases that were studied, its presence was demonstrated in some. One of the classical instances to be found in the literature of the detection of the tubercle bacilli in the lesions is the one unfolded by Kyrle.[15] His patient was a Serbian prisoner of war. The diagnosis was that of Boeck's sarcoid. Kyrle states that in the early lesions of this patient he was able to demonstrate the tubercle bacilli without

difficulty. In lesions of ten days' duration he found numerous acid-fast organisms; they were less numerous after twenty-one days, and on the thirty-sixth day, when the classical picture of the disease was fully developed, no tubercle bacilli were demonstrable.

Occasionally investigators have been able to show the tubercle bacilli in smears from nasal secretion.

Considering the large number of cases of sarcoid that have been studied and reported, the tubercle bacilli have shown an extraordinary elusiveness, escaping demonstration either from the lesion directly or from an inoculated animal indirectly. The tuberculin test has also shown an uncommon phenomenon of remaining mostly negative. This property of the skin of offering an inactive response to the tuberculin reaction has been termed tissue anergy.

I have looked through a variety of histological descriptions given by a number of authors, and while they might differ in unimportant words here and there, they seem to be in complete accord in the important features. There are certain essentials which give the histologic picture the accepted characteristics. The intrinsic structure is so typical that general pathologists, when shown sections of the sarcoid, often unhesitatingly pronounce them tuberculous. Inas much as most investigators agree about the inherent characteristics, without which a diagnosis of Boeck sarcoid cannot be made, I shall give the important highlights which go to form the representative pattern. These are largely molded after Stellwagon.

The foci of the new growth are separated from each other. Higher power magnification shows that the cells of the new growth are the type of epithelioid connective tissue cells, and that the tumor, as a rule, has its origin in the perivascular lymph spaces. The proliferated cells soon enclose the greatly dilated vessels with a compact cylindric mass. As proliferation increases and the foci take different shapes, though remaining still sharply circumscribed, the resemblance to epithelioid cells becomes more marked. The nuclei are large and vesicular, less deeply stained and show distinct nucleoli; the nuclei are sometimes multiple. The cell protoplasm is increased in amount and sends out prolongations in different directions. In a few instances giant cells of the sarcomatous type are found. Mitosis is scarcely anywhere to be detected. At the periphery are seen lymphocytes in varying numbers, few plasm cells, and scattered here and there are giant cells having many nuclei.

Some authors (Fox[16]) hesitate to recognize the giant cells to be true Langhans type, while others (Pinner) state that within

the epithelioid follicles occasional giant cells of the Langhans type are seen. Necrosis and caseation are absent, although single epithelioid cells may show some slight necrobiotic changes. Exudative features and polymorphonuclear cells are absent. The isolated islands are almost always devoid of a peripheral inflammatory zone and without central necrosis.

CASE REPORT

The patient was referred to the rhinolaryngological clinic of the New York Post-Graduate Hospital of Columbia University by the Skin and Cancer Unit for examination of the nose and larynx to determine the cause of hoarseness from which the patient had been suffering for the past two years. There had also been complaints concerning both ears.

She was a fairly well nourished colored female, aged 41 years, presenting the typical dermatological sarcoid lesions of the face and body, an analysis of which follow.

For convenient purposes of description I shall divide the upper respiratory tracts into the external as well as the internal part of the nose and mouth with their adjacent organs.

The external portion of the nose might be usefully subdivided into the upper part, or dorsum, and the lower part, or base. The dorsum remained within normal bounds. The base, on the other hand, was studded with round or ovoid tumorlike masses about the size of a pea, giving it an irregular, distorted outline. The pathologic lumps showed themselves as elevated protrusions in the center or tip of the nose, or hung down as inverted mounds on the free rims of the nares. Those that hung down had a more indigo bluish coloration than those occupying any other part of the body except the face (malar region). The posterior border of the nasal nares was also studded with elevated infiltrations.

In the interior portion of the nose upon the septum of the right side at about the junction of the quadrangular cartilage with the vomer was a rectangular pathologic mass composed of closely packed miliary infiltrations. In appearance it could be likened to a flattened-out surface of a raspberry. It was approximately 1 cm. long and 0.5 cm. wide. In the main its surface was unbroken, but in tiny isolated spots it was covered with a crust. The patient told me that every now and then blood trickled out of her nose, which might account for the presence of the crust. After I shrank the

tissues with adrenalin and cocaine solution, there appeared to be an outline of scar formation on the lower turbinate of the same side. The reason for the scar tissue was, of course, open to debate, despite the fact that it was practically opposite the infiltration of the septum just described. All these pathologic bodies alluded to were nontender and showed no evidence of ulceration. In the left side of the nose the septum was convex and upon the extreme convexity, about 1½ cm. from the anterior inferior spine, was a large indurated and infiltrated area, which, when touched with a probe, bled easily. While no biopsy of this particular area was made, its clinical characteristics were so clear, corresponding to those taken frequently from other portions of the respiratory tract for laboratory investigations, that I had no hesitancy in accepting it as part of the general pathology.

Apropos of the above findings, it might be of interest to give a short abstract of the paper by Fletcher and Novy. When the three cases are set in juxtaposition some fair evaluation perhaps can be reached. Fletcher's patient suffered from an interior nasal induration, from which she had nosebleed, and when she first presented herself for medical treatment there were no skin lesions, the dermatological lesions making their first appearance long after the nasal induration occurred. Novy, in his paper, states that his patient was seen December, 1933, when she suffered from nosebleeds, and that in May, 1934, when she was again seen because of nosebleeds, the patient had an eruption of several months' duration on one arm. Of the three, Fletcher's case is perhaps the only one in which it can be accepted with a certain degree of security that the nasal infiltrations preceded the appearance of the dermatological lesions. Novy's case is somewhat cloudy on this issue. The pathologic nasal patches in my patient were observed long after the patient had the skin eruptions. Still, a certain degree of common ground can be found in the three cases which suggests that possibly a nasal lesion did exist prior to the affection of the skin.

My patient was questioned many times at length about the early nosebleeds she had before she became aware of her skin troubles. Due to the length of time that has elapsed since she had first observed her cutaneous changes, her first responses were not entirely free from the suspicion that she was guessing. My patient unhesitatingly referred to her nosebleeds of years ago. In answer to my questions, she replied that they might have been prior to her first attack of skin troubles. I was particularly anxious to establish whether the nosebleeds or nose sores occurred before the cutaneous

infiltrations appeared. Though the patient was conscientious in her replies, the lapse of time mitigates unqualifiedly accepting her answers. Neither can the supposition that the nasal or laryngeal lesion antedated the skin efflorescence be ignored. Should these deductions be found to be correct, it can rightly be assumed that the portal of entry of the original infection was the upper respiratory tract system. Seiferth's case perhaps sheds some light on this particular point. The remains of the epiglottis of his case resembled that of my patient in some respects. Should the upper respiratory tract prove to be the portal of entry to the organism of this unfortunate disease, the first shock troops to uncover and attack this ailment in its incipiency will be the rhinolaryngologists.

On the lip of the right side of my patient there was an oval area 1 cm. long and about 5 mm. wide. Its color varied from light to dark blue, and it was raised above the surrounding tissue. It was covered with unbroken glistening mucous membrane. The size of this plaque was not always the same. During the two and one-half years in which the patient has been under my observation, I have seen the size alternately becoming smaller and larger. When the plaque was reduced in size the lip would feel quite comfortable, but when an increase of size occurred the patient experienced a constant burning, boring sensation. The discomfort became aggravated when the patient was eating or drinking. A constant urge to moisten the lips was present, which when done would only increase the discomfort. The description of the burning, boring sensations leads me to think that they are similar to those heard from patients suffering from herpes labialis. There were times when I had the impression that the color of the plaque was a deeper indigo blue than at other times. It is difficult to account for the different gradations of color, except that it often appeared to coincide with the menstrual period, indicating a probable endocrine imbalance. There was no blood vessel or vessels that could be recognized as such with the naked eye within the affected area. Since this patient was undergoing therapeutic experiments I preferred to defer the removal of this involved labial tissue for histologic examination. At the time the burning sensations would become manifest, the lips also would become puffed and enlarged, and at the disappearance of the disagreeable sensations the puffiness also disappeared. On the upper and lower lips immediately above and below the vermilion were single infiltrations varying in size from 1 to 1½ mm. The skin too was unbroken and the color unchanged, and it did not move over the infiltrations when an attempt to move it was made.

In the center of the hard palate, about 1 cm. from the left incisors, extending backward into the soft palate and uvula, an irregular chain of pinhead-size infiltrations was present. The surfaces of several were broken. Those with broken surfaces were located entirely in the hard palate. The break in the surface of the several infiltrations was unquestionably due to the friction engendered when particles of food rubbed over them during the process of eating, drinking, etc. The loss of substance may be looked upon as a purely mechanical, frictional factor from the outside and not inherent to the infiltration. The sinus pyriformis appeared normal.

The false cords of the larynx, particularly the left, were hypertrophied and showed numerous seedlike elevations. They encroached upon the free space during phonation. The true cords appeared to be free from pathology and readily responded in their physiologic movements. The chink, however, was not even during phonation, which was attributable to interference by the presence of pathology in the immediate neighborhood.

In the position of respiration there could be seen immediately beneath the vocal cords, i.e., in that part continuous with the thin lateral part of the conus elasticus, an abnormally thickened mass on each side protruding into the lumen of the trachea. Five tracheal rings could be counted through the laryngeal mirror. On the posterior wall of the trachea were numerous thickened pinhead spots which felt hard when touched with a probe. The patient had had dyspnea for several years and found it difficult to perform her daily chores without getting tired and out of breath.

The arytenoid cartilages were infiltrated and quite edematous. They were also somewhat nodular. The interarytenoid space was only slightly thickened, an interesting finding because in frank tuberculosis, whenever laryngeal involvement occurs, this space very often harbors a good part of the pathologic invasion. The aryepiglottic fold, as they leave the tips of the arytenoid cartilage and just about as they reach the epiglottis, appeared thin, soft and free from edema.

The entire epiglottis was thickened, giving the impression of being at least twice as thick as normally. There was considerable nodulation. The nodes were not uniform in size. Their surfaces were unbroken except at a small area at the free edge. On the lingual surface, lateral to the medial glosso-epiglottic fold on the right side, was a comparatively large, round tumorlike mass, filling the greater portion of the valecula, while on the left side the pathologic invasion

was smaller. Part of the projecting new growths were removed for microscopic examination. The laryngeal surface appeared to be sprayed with discrete miliary infiltrations. The epiglottic tubercle was twice its normal size. The glosso-epiglottic folds were normal. The mucous membrane covering the nodules in the valecula of the right side was strikingly thin and glistening.

A routine bronchoscopic examination was carried out by Dr. Paul Seager. I was present during the entire progress of the investigation and had the privilege of inspecting the tissues through the bronchoscopic tube. In addition to the subglottic and tracheal new growths already mentioned, there were also small tumorlike agglomerations deep in the trachea close to the bifurcation. Their location approximated the left anterolateral aspect of the windpipe. The bronchoscopic tube was passed beyond the bifurcation, but because of the length of time consumed in removing tissue for microscopic examination a thorough inspection of the smaller bronchi was not feasible. It was difficult to obtain a satisfactory amount of tissue from the deep portions of the trachea, because the new invading growth was hard and resisted cutting; it was only after much effort that a small section was finally procured for laboratory use. All of the growths thus far noted were of a fleshy appearance, and when not traumatized, were practically all covered with unbroken mucous membrane; in some parts the mucosa was shiny and glistening while in other parts it was dull and congested.

Three months afterward another bronchoscopic investigation was undertaken. This time the tube was introduced beyond the carina into the smaller bronchial tubes on the right as well as left side. The tissues were normal except as described in the foregoing paragraphs.

On the upper portions of the patient's legs and arms large patches of indurated tissue, having a rubbery feel to the examining finger were located. Most of these patches showed a marked reduction of pigment as compared to the surrounding normal skin. They were of irregular size and shape; ssome were as much as 4 or 5 cm. in diameter while others were only about 0.5 cm. In shape they were circular, ovoid or quite irregular. The trunk and neck were singularly free from any evidences of pathology.

Roentgenologic pictures as well as their interpretations were done in the Department of Roentgenology of the New York Post-Graduate Medical School and Hospital of Columbia University. Discussion was held at length between Dr. Meyer, the head of the department, and myself, over whether any evidence could be ob-

served in the roentgen pictures that might be construed as pathognomonic of sarcoid disease. After due consideration of this case coupled with a thorough search of records of two other patients, we came to the conclusion that there was insufficient characteristic evidence to make a diagnosis of sarcoidosis from radiographic pictures only. Chest examination of the thorax showed the total heart area to be relatively small (the transverse diameter being fully 4.5 cm. below the average in normal persons) though of normal contour.

There were lymphomatous masses along the tracheobronchial tract, the larger and more circumscribed one of these being about 4 cm. in diameter and protruding just above and to the right of the aortic arch. There was, however, further marked increase in density, again lymphomatous, at the hilus and the root, with moderate secondary root branch and central bronchial thickening. The lesion was entirely central in origin and extent, with the peripheral lung fields comparatively clear.

Except for a moderate generalized vascularity there was no evidence of recent parenchymatous infiltration or pleural involvement.

The hands showed circumscribed pea-sized areas of osteoporosis in some of the phalanges, mainly at the head of the proximal phalanges of the left fourth and fifth fingers. The cancellous structures of the bones of both hands appeared somewhat coarsened, and radioparent.

Examination of the feet showed a number of very small areas of rarefaction in the phalanges similar to the changes noted in the hands. There was no pathology in the long bones.

The mandible showed a large, circular area of rarefaction, situated in about the center of the horizontal portion of the bone.

I have not seen any previous reports of a circumscribed osteoporosis of the mandible ascribed to this ailment. Within the past decade or more pathology of the oral cavity and the structures forming this space have been subjected to intensive study and more detailed knowledge of the disease described here as affecting this cavity and its structures might be of especial interest to those specializing in the study of the mandible.

Microscopy:

The Department of Pathology of the New York Post-Graduate Hospital reported the following on microscopic examination of tissue sections: "Tissue examined was taken from epiglottis, larynx, and

subglottis as well as small pieces taken from the trachea near its bifurcation.

"Sections from all the tissue show the same reaction. Beneath the epithelial covering the stroma is made up of closely packed epithelioid cells partly in outlined tubercles, a few of which contain giant cells of the Langhans type. There is no caseation.

"Diagnosis: Non-caseating tubercles suggesting sarcoid."

Examination of skin tissue taken from the tragus of the ear was as follows: "The sections have a covering of stratified squamous epithelium. At one point only a few layers of epithelium remain on the surface and this shows exfoliation. The underlying stroma contains numerous hair follicles and is marked by several oval areas of epithelioid cells which replace the connective tissue stroma. At the margin of these areas there is a small amount of lymphocytic infiltration. No giant cells associated with the tubercles are seen in the sections examined."

The report from Department of Pathology of the Skin and Cancer Unit of skin tissue taken from another part of the body of this patient stated: "The epidermis is thin and the basal layer deeply pigmented. Throughout the cutis are many sharply demarcated nodules composed of epithelioid cells and fibroblasts. A few round cells occur as well as multinucleated cells. Marked vacuolization and considerable granular degeneration of the cellular infiltrate is noted."

The above three microscopic reports, one on tissue obtained from the upper respiratory tract, and two on the skin show in the main identical architecture.

Examination of the blood showed: hemoglobin, 72; erythrocytes, 3,850,000; leucocytes, 4,350. The differential white cell count of 200 cells showed: polymorphonuclear neutrophiles, 47 per cent; lymphocytes, 40 per cent; eosinophiles, 3; basophiles, 0.5; monocytes, 9.5. The red cells showed some achromia.

From time to time the patient was subjected to the tuberculin tests of varying lengths and strengths from 1:1,000,000 to 1:0. The results were always negative. The tests consisted of the Mantoux, von Pirquet, and the patch. The patch test was undiluted. The materials used were human, bovine and avian tuberculin. Pus obtained from an apparently completely closed sack located in the face was cultured for tubercle bacillus and proved to be negative.

I was assisted in the tuberculin patch test by Dr. Maurice Grozin,[17] as described by him.

As previously mentioned with diascopy, minute, dirty, yellowish foci were seen, as observed in lupus vulgaris. In my patient diascopic pressure was applied only on the face with positive results. The area which responded positively to the test had the appearance of comedones before diascopy. The cheeks had undergone many different forms of therapeutic attention. The last, and verified, form was the application of electrocoagulation to the indurated tubercles. Then for several months after the patient came under my care, it was observed that localized regions in the cheeks puffed up, becoming sensitive, and then broke, evacuating pus through the broken surface. Normal healing followed. Only a short period elapsed before another infection occurred, and abscess followed abscess at frequent intervals. These most often broke and emptied spontaneously. It was difficult to establish the reason for the frequent occurrence of the purulent pockets because the patient came to the out-patient department and therefore was not under constant medical surveillance.

In due course, however, a careful check brought out the following: The patient's face perspired freely. The perspiration had a slippery, oily feel to the touch of the fingers. There were numerous spots resembling the commonly called blackhead in many parts of the face. Many of the comedo-like areas when subjected to pressure yielded a plug of dried sebaceous matter. In the cheeks, where electrocoagulation was used to destroy the pathologic infiltrations, the comedo-like bodies which accumulated from time to time were infected secondarily, and an abscess formed, though the abscess was singularly accompanied with little pain. One of these abscesses, which possessed an apparently unbroken sack, was carefully aspirated with sterile needle and syringe and cultured. Staphylococcus aureus was obtained. All search for tubercle bacilli was fruitless.

SUMMARY

As far as I have been able to ascertain this paper is the first to describe a clinical multiple benign sarcoid of the Boeck type of the entire upper respiratory organs. To increase its value a description of other parts of the body affected is included. A short discussion of the disease is likewise deemed of importance, and therefore incorporated. There is much to sustain the assumption that the upper respiratory tract might be the portal of entry to the organism

responsible for this malady. A discussion of the laryngeal affection of this disease was published in 1940.[17]

Fortified with a more intimate survey of the clinicopathologic manifestation of this disease, the rhinolaryngologist can manifestly play an ever-increasing roll in detecting early morbid changes in some part of the upper respiratory tube, and thus perhaps head off numerous ultimate complications.

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XLII

MEDICAL RESEARCH AND WAR PROBLEMS*

FOREWARNED IS FOREARMED

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DETROIT

Those of us who served in the last World War and who now live in defense industrial areas are daily reminded of many advances which medical research has made in the last two decades, as compared with our inadequate status in 1917 to 1918.

December's compelling events placed an "immediate, and tremendous responsibility on the medical profession,"[1] and the profession was not found off guard. We in the midwest industrial region are faced with the grim reality that since this present world upheaval is a war of production, the cities in which its producers live are potential military targets.

Yet our research laboratories have not limited their studies to changed methods of treating casualties from bullets and bombs. Military charts are constant reminders that a higher mortality rate is recorded by the spread of disease in temporarily congested areas.

The assembling of army recruits or industrial workers from different parts of the country and different environments into close contact introduces a new condition which favors the transmission of disease. It is inevitable that such an assembly, with varying degrees of susceptibility to different infections, will bring into the community an infinite assortment of those pathogens which are indigenous to the respective home communities of the migrants.

Preventive vaccines have practically conquered the old disease perils of diphtheria, dysentery, tetanus, typhoid fever, and smallpox. As reported by Mosher,[2] "it is a striking fact that after Dunkirk there was practically no tetanus in the English troops, due to the routine use of tetanus toxine during the training of the men." Our medical officers were hampered in some sections by public disapproval of the use of this vaccine; but, according to the

*Presented before the section meeting of the American Laryngological, Rhinological and Otological Society, Jan. 21, 1942, at St. Louis.

Post Surgeon's staff at Fort Custer,[3] "it is a matter of record that the Army of the United States does now require universal immunization for tetanus administered in three doses of the liquid toxoid with three weeks interval, and reinforced by a further dose, once a year, on entering a theatre of war or upon incurring an injury."

Although there is, as yet, no proved method of preventing influenza nor its pneumonitic complications, recent reports from serologic and chemotherapeutic investigators foretell a marked decrease in the mortality rate of these diseases which proved to be the major problems of World War I.

As listed by Pepper[4] of the National Research Council, the following six diseases caused 88 per cent of the total of 34,855 deaths among our enlisted men and officers: influenza, 16,571, or 7.41 per thousand; lobar pneumonia, 5,787, or 2.59 per thousand; bronchopneumonia, 4,143, or 1.85 per thousand; measles, 1,987, or 0.89 per thousand; tuberculosis, 1,457, or 0.65 per thousand; meningococci meningitis, 986, or 0.44 per thousand.

Since the remaining 12 per cent consisted of so many "small increments", its consideration is of no great interest to medical scientists other than to intensify their efforts to solve the graver problems of the three major respiratory infections.

The specific prophylaxis and treatment of pulmonary infections are based on definite etiologic classification and diagnosis. For primary pneumococcic pneumonia (usually lobar) the National Research Council[5] recommends chemotherapy as the method and sulfathiazole as the drug of choice in all cases. They have found this therapy is not only "effective against all types of pneumococci", but is "also applicable to treatment of most bacterial pneumonia other than pneumococcic."

Conversely, with secondary pneumonia: "The possible value of chemotherapy with sulfanilamide and its derivatives in the prevention of bacterial pneumonia complicating such diseases as influenza and measles is not known at present. Specific recommendations are, therefore, not justified." This therapy is of "no demonstrated value for many patients with bronchopneumonia of indeterminate (virus?) etiology; it is also of no demonstrated value against pulmonary infections with Str. viridans or Haemophilus influenzae."

On the other hand, sulfathiazole is recommended in those cases of secondary pneumonia in which pneumococci, hemolytic streptococci, staphylococci or Friedländer's bacilli are found and believed

to be of etiologic significance. The dosage and the precautions are the same as in cases of primary pneumonia.

In his investigations of many of the aspects of the problem of epidemic influenza, Francis[6] found that there are fundamental differences in the causative agents of epidemic recurrences and that these differences are of primary significance for an understanding of immunity and the development of prophylactic measures. But the basic problem confronting the investigator of epidemic influenza is primarily that of explaining its numerous recurrences.

Confusion in diagnosis has prevailed despite the fact that symptomatology of epidemic influenza has, throughout its history, presented "an amazing uniformity." The disease is characteristically epidemic; develops quickly; spreads rapidly—by air, by contact and by travel—to as much as 30 to 50 per cent of the population; terminates abruptly; and is seldom fatal unless complicated by a pulmonary infection.

Horsfall[7] attributed this confusion in diagnosis largely to the fact that acute upper respiratory diseases tend to resemble each other very closely, hence their classification on clinical grounds has not been very successful. There is "no sharp line between the symptomatology of a severe common cold, sporadic grippe, and influenza." Even among cases in which the etiology of the disease was determined, no objective signs or symptoms were discovered. Also the tendency of influenza toward epidemicity is of no great help since epidemics of influenza usually occur when there are also marked increases in the incidence of other respiratory conditions.

It is now known that this epidemic disease may be due to any one of at least three infectious agents. Two of the causes of influenza are known to be viruses. The first, now designated influenza A virus, was described by Smith, Andrewes, and Laidlaw,[8] in 1933; the second, influenza B virus, was described independently and almost simultaneously by Magill[9] and Francis[10] in 1940. The evidence that at least a third agent exists is based upon the fact, that, during the past year, of more than 850 cases of influenza studied by Horsfall,[7] approximately 30 per cent showed no evidence of infection by either the influenza A or the influenza B virus.

That the systemic defenses, exemplified by circulating antibodies, do not fully control susceptibility to infection in the upper respiratory tract, was observed by Francis[11] in a series of experimental studies. "At the onset of an attack of epidemic influenza, the blood of a majority of patients contains sufficient antibody to

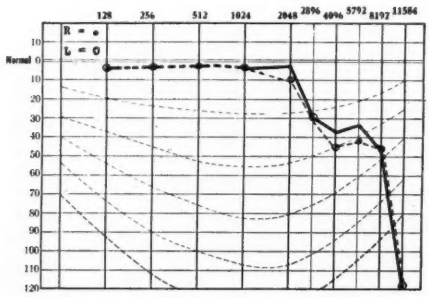

Fig. 1.—(Jan. 11, 1942) C.L.L., aged 29 years, has been flying eight years and has had 1,000 hours in the air. The highest altitude at which he has flown is 25,000 feet. He has used no ear protection except when flying 1,000 horsepower open type planes. While making a flight he noticed some difficulty with radio reception and before reaching his destination, it was necessary to turn on the full radio volume in order to hear landing instructions. As soon as the plane reached the runway, the broken antenna came in contact with the ground for 100 to 150 feet before the earphones could be removed. This extremely loud static noice caused great impairment of hearing for several days. The loss of higher frequencies evidently was due to this accident. It was about two weeks after the accident before hearing returned for easy conversation.

protect experimental animals against many lethal doses of influenza virus." In some instances it is equivalent to that reached by other "patients in convalescence when antibody formation and immunity are at their height."

On the other hand, it is not uncommon to find individuals who possess little or no demonstrable antibody, but escape infection although intimately exposed. Influenza virus selectively attacks the cells of the nasal respiratory epithelium, a "luxuriant superficial epithelium separated by a cribriform membrane from the underlying blood vessels." Francis found reason to believe that the virus could gain admission to the epithelial cells without coming into contact with antibodies of the circulating blood.

Hence a variation in functional activity of the cells is of fundamental importance in maintaining the resistance of the respiratory epithelium in infection. Of equal significance is the evidence disclosed during the studies of nasal secretions that these contain a substance capable of inactivating the virus of epidemic influenza. The inactivating agent is indicated to be an antibody of the physiologic, natural type. By its presence at the surface it has an

opportunity to exert its effect immediately upon the arrival of an infectious agent.

"These two manifestations of local immunity, one cellular, one serologic, may conceivably complement one another. Each presents a method by which an injurious agent reaching the tissue of predilection may be combatted." Destruction of the respiratory epithelium is not prevented by antibodies in the blood. Since this tissue is superficial, it is exposed to infection in such a manner that defense to be efficient must depend primarily upon its effect on the nasal mechanisms of resistance.

The treatment outlined for the U. S. Army[12] by the Surgeon General's Commission on influenza is summarized as follows: "The treatment in uncomplicated cases of influenza is at present symptomatic. There is no evidence that chemotherapy influences the primary virus disease. The patient should be kept warm in bed for 48 hours after his temperature returns to normal. Cold air frequently aggravates the irritation of the respiratory passages. Steam inhalations and soothing cough mixtures with codeine often relieve a persistent distressing cough. Mild antipyretics and sedatives may be used, but narcotics other than codeine are generally contraindicated. Fluids should be given to 3,000 c.c. or more daily. The bowels should be regulated when necessary with mild cathartics or enemas. A soft diet can be given as soon as the patient's appetite will tolerate it.

The patient should resume his normal activities gradually. When secondary bacterial invaders are prevalent, protective isolation for a week or more after recovery should be given. Any unexplained rise of temperature or a return of fever should be interpreted as the onset of a pulmonary disease due to pathogenic bacteria. Under these conditions adequate measures for the diagnosis of pneumonia and the determination of the infecting bacterial agent should be instituted. Chemotherapy or other available types of therapy directed against the invading bacterium should be used."

The constructive orders issued by the Surgeon General of World War II dealing with the treatment of influenza present a contrast to the state of unpreparedness in 1917-18.

Another recurring problem of war is the transmission of infection to war wounds, one of the chief offending pathogens being the hemolytic streptococcus. On the basis of many investigations, it was shown that no single infectious agent was responsible for

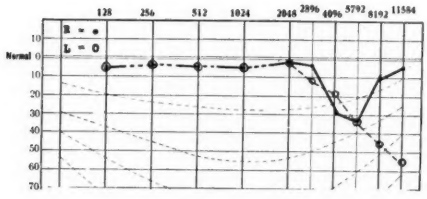

Fig. 2.—(Jan. 11, 1942) J.R.H., aged 33 years, has been flying for 10 years and has had 3,000 hours in the air. The highest altitude at which he has flown is 18,000 feet. He has used cotton in his ears occasionally. In 1918 he had a mastoid operation on his right ear and in 1937 had two spontaneous ruptures in the right ear drum from dive-bombing. These were very painful but there was no bleeding. Dive-bombing was started at 12,000 feet downward to 3,500 feet when the ear drum ruptured. Hearing was greatly impaired for three weeks.

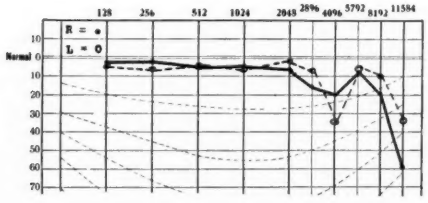

Fig. 3.—(Jan. 11, 1942) C.L.P., aged 24 years, has been flying for two years and has had 330 hours in the air. He has flown as high as 18,000 feet in an open cockpit without oxygen and has experienced 29,000 feet altitude in a decompression chamber. Plugs of cotton have been used occasionally in the ears. Most of his flying has been done with the "deep low-noised plane" of the open type. He has used a light helmet most of the time.

more deaths during the last war. Yet, as Rantz[13] has pointed out, it has been possible only recently to classify hemolytic streptococci accurately, and to study the epidemiologic aspects of the infections produced by them.

From his observations on the "carrier state" in the San Francisco area, he concluded that "as many as 50 per cent of normal persons in whom the tonsils are intact may harbor hemolytic streptococci of group A during certain seasons of the year"; and that these organisms are "normal inhabitants of the human nose and throat."

They were isolated from 31.4 per cent of wounds sustained in the evacuation of Dunkirk, France, and examined after return to England. Miles and associates[14] have shown that hemolytic streptococci are seldom found in the wounds of injured persons on admission to the hospital; but that they soon appear and increase in frequency during the first two to three weeks of hospitalization. The reason given is that, in the vast majority of wounds infected by hemolytic streptococci, the infection has its source in the nasopharynx of the patient, his attendants or his fellow patients.

In furtherance of this aspect of wound infection by carrier transmission, these collaborators noted that many infections in a ward or hospital are due to streptococci of the same type. Also they observed the spread of a single type within a ward following its introduction by an infected person. The new type appeared, not only in previously uninfected wounds, but in infected ones, with an aggravation of symptoms. Several attendants became carriers of the same type, thus accelerating the transmission of the infection.

Modern research has demonstrated that expired droplets with a diameter of 0.1 mm. or less are so small, or quickly become so by evaporation, that they remain suspended in the air indefinitely and may be air-borne for considerable distance. If these fine particles remain viable and virulent, persons susceptible to the particular air-borne pathogen may become infected some distance from the source of infection. The viability of non-sporing pathogens outside the animal body is highly significant in this method of transmission; the streptococcus and pneumococcus; the diphtheria and tubercle bacilli, also the viruses of smallpox, chickenpox and measles are hardy on both a bacteriologic and an epidemiologic basis, if they are protected from light. Since dust-covered droplets resist light, every precaution should be taken to prevent the accumulation or dissemination of dust.

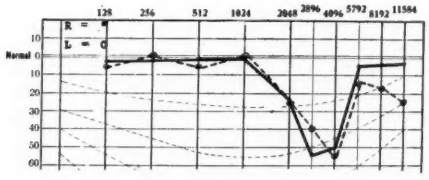

Fig. 4.—(Jan. 7, 1942) A.G.T., aged 33 years, has been flying for 10 years and has had 1,500 hours in the air. He has flown at 16,000 feet and has experienced 40,000 feet with oxygen in a decompression chamber. He has never used protection in the ears. No notices fatigue from the vibration, having done dive-bombing, gunning, etc. He has "no ear trouble".

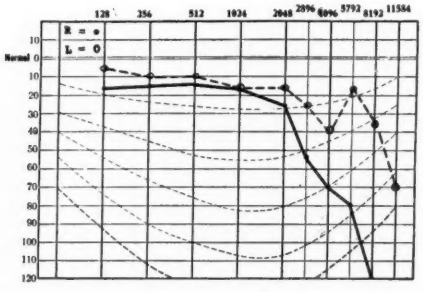

Fig. 5.—(Jan. 11, 1942) E. N., aged 51 years, has been flying for 25 years and has had 8,000 hours in the air. He has flown at high as 25,000 feet. During the last war he flew 350 horsepower biplanes (Liberty and Curtiss) and has flown some 1,000 horsepower planes. He was in Naval Aviation from 1916 to 1922 and re-enlisted in 1941. At present he is not flying due to the age limit. He has never used ear protectors and flew many hours without his helmet.

More specific means of preventing air-borne infections are the masking of suspected carriers; good, natural ventilation; and the use of ultra-violet radiation. Blankets and bed linens may receive the discharges from wounds or infective droplets from the respiratory tract. Blankets should be encased in easily washable covers, and bed linens should be soaked in 2 per cent lysol.

When conditions are available for a precise study of an epidemic of infections caused by hemolytic streptococci Group A, Rantz[13] advocated his simplified method of serologic typing. Since it has been shown that immunity to the hemolytic streptococcus is type specific, there will be found in any community that a limited number of types will be responsible for the bulk of infections. Furthermore, since only a small number of injured persons will harbor these organisms on admission to the hospital, "it should be a simple matter, by frequent cultures of wounds and of the throats of patients and attendants to establish the mechanism by which infection is spread and to devise means for its control." An important application of the Rantz method of typing may arise if epidemics of postinfluenzal pneumonia caused by hemolytic streptococci again become common in military and industrial centers.

Aside from the field of infectious conditions, there is a newer problem born with mechanized warfare, which involves many aspects of physical and psychologic defections resulting from occupational noise. The hazards of noise are not limited to our military operations, but are experienced, likewise, by our vast civilian army engaged in producing the munitions of war.

Although industrial workers are seldom subjected to high, explosive noises, yet they endure a daily grind of irritating noises of various intensities and pressures which often have an increasingly harmful effect upon the central nervous system. This is especially true of the skilled mental worker, the nervous factory type of worker, the neurotics and those who are otherwise ill.

Muscular and mental fatigue means a dangerous drop in hourly output of equipment vitally necessary to the safety of our armed forces. To a great extent the very life of our nation depends upon the health, morale and efficiency of our industrial workers, yet research in this field is of comparatively recent date.

The damage to mankind resulting from noise has not as yet been adequately investigated, according to Knudsen.[15] It is well known that prolonged exposure to the ordinary noises of many occupations—riveting, forging, weaving, and hundreds of other types

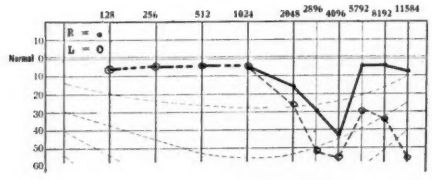

Fig. 6.—(Jan. 11, 1942) C.L.C., aged 37 years, has been flying for 22 years and has had 7,000 hours in the air. He has done test flying, stunt flying, dive-bombing and gunning and has flown as high as 38,000 feet, beginning to use oxygen at 12,000 feet. In the decompression chamber he has experienced 18,000 feet without oxygen and 30,000 feet with oxygen. At one time he flew for one or two hours at Mexico City at 24,000 feet without oxygen. While flying 500 to 1,000 horsepower open type planes, he has used cotton packed in his ears.

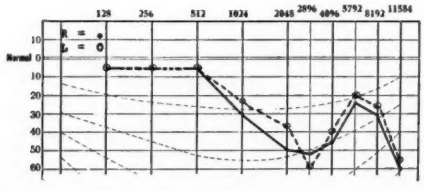

Fig. 7.—(Jan. 7, 1942) L.C.Y., aged 41 years, has been flying for 17 years and has had 4,900 hours in the air. He has flown as high as 40,000 feet with oxygen. He has flown all types of heavy motored planes, such as heavy bombers, carriers and patrol planes since 1925 and has seen duty in Panama and various parts of the world. He states that his first audiogram, made in 1937, showed similar loss of higher frequencies as does the present audiogram in which loss now is more pronounced.

of factory employment—results in premature loss of hearing; and that even short exposure to explosive sounds often results in serious injury to the auditory mechanism. But accurate data are lacking in respect to the degree of damage to the human mechanism.

In their review of a series of investigations of the effects of noise on the auditory mechanism of experimental animals, McCord, Teal, and Witheridge[16] cited several authors who reported that experiments on animals show very definitely that high-pitched sounds of various kinds and low-pitched sounds under certain conditions of resonance produce degenerative changes in the organ of Corti.

Other experiments with animals have shown a tendency for hearing to be impaired more by vibrations transmitted through the bony structure of the body to the ear than by air-borne vibrations coming through the external ear.

Microscopic examination of animals subjected to definite sound frequencies has shown that the section of the cochlea most seriously affected is that which is believed to be resonant to the test frequency. More recently, despite the histologic difficulties which arise when dealing with human material, lesions in the basal turn have been demonstrated in boilermakers and others who are subjected to loud noises of high frequency for some years. These workers become deaf to high tones.

By correlating the range of frequencies over which the patient was deaf with the position of the cochlear injury, examined histologically after death, these regions of the human basilar membrane which are resonant to frequencies from 2,000 cycles per second upward have been roughly located.

A comparison of contemporary chart records of human hearing impairment indicates that industrial workers, aviators, and others exposed for a considerable period of time to particular frequencies become deaf to those frequencies, and in the majority of cases deficiency appears first in the high frequency range. The lower part of the organ of Corti, which is the area concerned with the perception of the higher frequencies of sound, appears to be especially vulnerable, either because of the small amount of blood supply to that area or because it is the most delicate and highly developed part of the organ.

Recordings from audiometric examinations of 19 Naval aviators have been obtained by the author before and after certain periods of flying. Most of these audiograms show a severe loss of

hearing of frequencies in the upper register above 2,000 cycles. With two exceptions out of 19 recordings, acuity for low tones is not considerably impaired. These audiograms are in definite accord with the deductive reasoning of Bunch.[17] From his comparative rating of the intensity level of noise in a boiler-shop at about 100 decibels and that from an airplane motor at about 110 decibels, he made the following deduction: "If continuous exposure to the noise in a boiler-shop results in diminished hearing, it is logical to expect that persons who are exposed to the noise of airplane motors will also become hard of hearing and that the louder noise will produce hearing losses more frequently and more quickly."

CASE 1.—Sudden hearing loss is noted in the audiogram of C. L. L. (Fig. 1), who, while making a flight, noticed some difficulty with radio reception. Before reaching his destination, he found it necessary to turn on full radio volume in order to hear landing instructions. When the plane reached the runway, the broken antenna came in contact with the ground for 100 to 150 feet before the ear-phones could be removed. This extremely loud static noise caused great impairment of hearing for several days, and permanent loss of hearing for the higher frequencies. It was about two weeks before hearing returned for easy conversation.

CASE 2.—Spontaneous ruptures of the right ear drum while dive-bombing in 1937 are recorded in the gram data for J. R. H., aged 33 years, who has been flying for 10 years and who has had 3,000 hours in the air. The highest altitude at which he has flown is 18,000 feet. He has used cotton in the ears occasionally. In 1918 he had mastoid operation on right ear. The ruptures were very painful, with no bleeding. Dive-bombing was started at 12,000 feet downward to 3,500 feet when the ear drum ruptured. Hearing was greatly impaired for three weeks although, paradoxically, the right ear, at present, shows better hearing for high frequencies than the left.

CASE 3.—C. L. P., aged 24 years, has been flying for two years and has had 330 hours in the air. The highest altitude he has experienced is 29,000 feet with oxygen, tested in decompression chamber, and in an open cockpit plane at 18,000 feet without oxygen. He has used plugs of cotton occasionally in the ears. Most of his flying has been done with the "deep, low-noise plane, open type." He used a light helmet most of the time, similar to type worn by motorcycle officers. There is marked loss of higher frequencies.

CASE 4.—A. G. T., aged 33 years, has been flying 10 years and has had 1,500 hours in the air. He voluntarily mentioned "fatigue from vibrations" with open type planes. He does dive-bombing, gunning, etc., but has "noticed no ear trouble."

COMMENT

Since each of the 19 charts presents such a variety of individual considerations concerning age, hours in the air, altitude, number of years of flying, degree of hearing loss for higher frequencies, it is difficult to formulate uniform conclusions. Further studies of im-

proved helmets and screening devices for higher frequencies are being made by the author.

According to Ceres,[18] the ear, nose and throat become an important part of the examination of flight students who volunteer for the special type of flight, as for instance, the fighter (interceptor, pursuit pilot, etc.). "These pilots will have to fly high to get above the bombers to do their work and that means above 30,000 feet, where atmospheric conditions play their part." Various susceptibilities to atmospheric changes may be indicated by anoxemia, air-sickness, neurocirculatory asthenia and phases of psychologic and physiologic effects of altitude on the nervous, respiratory and circulatory systems.

Ceres further reported that research in aviation medicine is now directed more to the physiologic maintenance of the flyer, his oxygen and oxygen equipment, and the problem of fatigue and its prevention. In this connection, Murphy and Barmack[19] are conducting experimental tests to determine whether certain drugs can be used to counteract "deleterious effects" produced on aviators flying at high altitudes. Military ethics obviously do not permit publication of the details but "successful results are confidently expected."

This attitude of confidence is evidenced by many reports from individual workers in medical research. In various fields a considerable number of our state and county medical societies have been undertaking the education of the lay public in health betterment. These activities demonstrate original methods and emphasize again the "importance of free and uninhibited enterprise in an independent medical profession."

"Education is undergoing profound changes together with all economic and sociologic phases of modern life. If these changes are constructive and to the benefit of humanity individually and collectively, the medical profession . . . may well apply the constructive ideas . . . in promoting that which is good." These constructive changes in modern education were emphasized by Shurly[20] with particular reference to handicapped children in relation to otolaryngology. The failure of early diagnosis and the neglect of treatment of the school child is responsible for a large proportion of the rejection of approximately 1,000,000 of the 2,000,000 men called in the recent draft, and pronounced unfit for military service. Under the Selective Training and Service Act of 1940, these men were disqualified because of physical, mental or educational reasons.

The individual in most instances was not aware of his physical defect. Certain of these defects are curable and their correction should become a matter of immediate concern in that the individual so affected may become a healthier, happier person. Certain of the defects are not curable, but the individual so affected will profit from proper medical advice toward the end of maintaining his economic status.[21]

From December, 1941, and onward to the termination of this mighty conflict, the life forces of our nation will be keyed to the grim realities of war. An outraged people will be united in a vast urge toward the destruction of our enemies. The pooled wealth of our country will now, as never before, be poured out in a concerted endeavor to gain a victory over the evil forces approaching our shores. Whatever the final verdict, the sad fact remains that no permanent good is achieved in the treacherous politics of war.

But we, who follow the teachings of Hippocrates, find recompense in the fitting words of Cannon:[22] "The conquest of disease, it should be remembered, is a permanent conquest."

662 Fisher Bldg.

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XLIII

CANCER OF THE LARYNX*

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AND

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There can be little disagreement with the statement that malignant disease is definitely increasing. Whether this increase is absolute or relative is a question which is still unanswered, but the appalling fact remains as an obstacle to the progress of medicine. It is true that heart disease continues as the largest cause of death in this country, but cancer now occupies second place in that grim listing.

Of all malignancies, about 4 per cent occur in the larynx and adequate warning is usually present, but unfortunately this warning is too often pathetically ignored. Here, unrecognized early carcinoma is the more tragic when it is considered that the early lesion offers a larger percentage of cures than carcinoma occurring in any other organ of the body.

Clerf states that 40 per cent of the cases of cancer of the larynx are so far advanced when first seen by the laryngologist that no type of surgery can be offered them (at that time). Certainly something can be done to alter this deplorable situation; and it does not appear too sanguine to suggest that this could be achieved by comparatively simple means. This would involve the cooperation of three groups. The laryngologist, as the member of the movement with the gravest of the responsibilities, should so spread the gospel of early recognition of carcinoma of the larynx that all coming within his sphere will learn to recognize and respect its symptoms. The general practitioner, having been apprised of these symptoms, should lose no time in urging laryngoscopic examination when such symptoms are present. And finally, the public at large should be made to develop its present state of cancer consciousness

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*Read before the Southern Section of the American Laryngological, Rhinological and Otolaryngological Society, Atlanta, Ga.

to a higher degree to the end that it will seek early and frequent examinations

Cancer of the vocal cord is a strictly localized disease which in its early stages spreads very slowly on account of the sparse lymphatic distribution at the anterior half of the larynx. Persistent hoarseness should make us suspicious of cancer of the larynx, but this does not imply that every case of hoarseness, or every lesion found within the larynx, is carcinoma. Tuberculosis and syphilis are frequently confused with cancer and must always be eliminated. Surgery of the larynx should never be attempted without first performing a biopsy to establish and verify a diagnosis. In innumerable biopsies we have not seen any deleterious results consequent on this procedure. The clinical picture must be studied carefully and if it is suspicious of carcinoma, repeated biopsies may be indicated. Recently we had occasion to see such a case which clinically appeared to be carcinoma. However, the pathologic report was chronic inflammation. It was not until the third biopsy was performed that we were able to obtain a report of malignancy. Had we accepted the first report the patient undoubtedly would have returned home feeling secure, only to be informed at a later date that the condition had probably progressed beyond the stage where surgery could be of any help. Carcinomatous lesions occurring in the larynx are necessarily seen in various stages of development and the method of operative approach advocated to eradicate the malignancy varies accordingly. On this basis we feel that an arbitrary division of these cases into four groups will simplify the classification.

The first group represents the earliest and the most definitely limited of all the lesions encountered. In it are placed carcinomas confined to a small portion of the surface of one vocal cord. The tumor may be only slightly larger than a millet seed or it may be as large as a matchhead or a pea, but it is essential that normal tissue be observed on all sides. Such a lesion is generally seen on the free border of the cord, somewhere near the junction of the anterior and middle thirds. From these qualifications, it may be readily concluded that this group includes only a very special type of growth which unfortunately is not seen often enough because of delay in laryngological consultation. When, however, it is encountered, much can be offered. Cure of the disease without mutilation and usually without the necessity of occupational or social readjustment can be promised with only reasonable reservation.

We believe that with suspension laryngoscopy, growths fulfilling these requirements can be completely and successfully removed

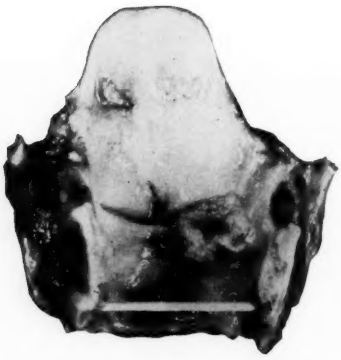

Fig. 1.—View of larynx, showing carcinoma involving entire vocal cord and extending into subglottic region.

Fig. 2.—Carcinoma showing involvement of ventricular band and base of epiglottis.

perorally by intralaryngeal dissection, following which electrocoagulation of the base may be employed as added insurance against recurrence. This method affords an adequate approach to the larynx; it is simple and safe in most cases; it is as accurate as removal by means of laryngofissure; it is followed by very little postoperative reaction; and the vocal end result is superior to that obtained by any other method of operation for carcinoma of the larynx. In addition, the continuity of the thyroid cartilage is preserved.

In advocating this approach, it is realized that many laryngologists are unalterably opposed to any form of intralaryngeal surgery in carcinoma of the larynx. When the late R. C. Lynch first reported his results by intralaryngeal dissection under suspension laryngoscopy for early carcinoma of the larynx, there were many who doubted the wisdom of his judgment and questioned the final results. Figi, in a recent publication, refers to this report and frankly admits that he was among the skeptics. "However," he states, "after cautiously using it and observing results during the course of a number of years, it can be heartily endorsed for use in a carefully selected group of cases." We wish to emphasize the last part of the quotation, "a carefully selected group of cases." If this is not rigidly adhered to, the operation is doomed to failure.

Suspension laryngoscopy is the only method of direct laryngoscopy which, besides providing satisfactory visualization, makes it possible to use both hands in intralaryngeal manipulations; and bimanual surgery must be employed for the successful extirpation of malignant lesions of the vocal cords.

When the larynx has been exposed, the tumor is grasped with a laryngeal fixation forceps and traction is made toward the median line. Traction toward the median line has a double purpose. It produces tension on the cord, making the incisions easier; and more important, it provides a maximum view of the lateral extent of the cord which is necessary if a wide margin of normal tissue is to be removed together with the neoplasm. The amount of tissue to be removed having been decided upon, a sharp laryngeal knife is employed for the excision. This is immediately followed by electrocoagulation which besides providing insurance against recurrence as has been previously mentioned, helps to control hemorrhage. If undue trauma has been avoided, the postoperative reaction should be negligible.

Formerly it was necessary to use chloroform anesthesia if electrocoagulation was to be employed. This was one of the objections

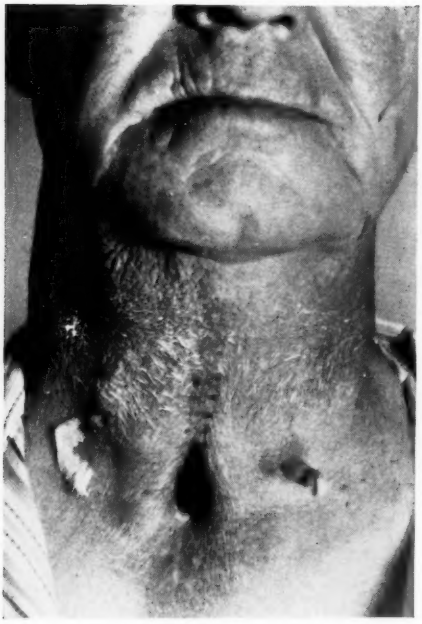

Fig. 3.—Appearance of wound on seventh postoperative day with drains still in place.

to this method of operation. Since the developent of pentothal sodium, this objection has been removed. Pentothal sodium is reasonably safe and produces sufficient relaxation for the proper performance of suspension laryngoscopy.

While suspension laryngoscopy is a comparatively simple procedure in most cases, it must not be supposed that it can be accomplished with ease at all times. An "Andy Gump" chin or a short, stout neck certainly increases the difficulties and in some cases it is impossible to expose the anterior commissure because of some anatomical peculiarity. Should these circumstances offer sufficient obstacles to a completely adequate exposure of the lesion, laryngofissure should be performed. Twenty-six cases have been operated upon by this method with excellent results. Fourteen patients are living and well in from five to 13 years and nine patients are living and well in from one to five years. One patient

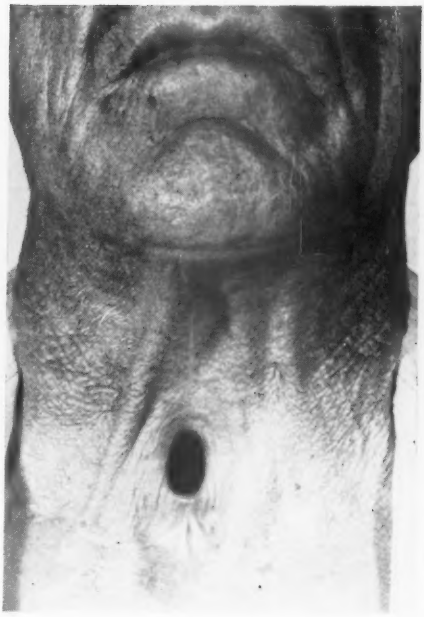

Fig. 4.—Complete healing of wound two weeks after operation.

lived three and another 11 years with recurrence and death; one patient lived six years, dying from pneumonia.

Until a few years ago we adhered strictly to the conservative and the radical methods of managing laryngeal carcinomas. There was no middle ground between these extremes. The conservative method referred to intralaryngeal dissection under suspension laryngoscopy; the radical method meant complete laryngectomy. It was realized more and more that there was a very definite place for an operative procedure that would fill that great gap existing between the conservative and the radical operations. The answer to this was laryngofissure and now there are few more appreciative of the merits of this operation than we, for its value is unsurpassed in those cases in which the lesion is too extensive for intralaryngeal dissection and yet not quite extensive enough for laryngectomy. The results obtained are excellent and the voice as a rule is good. In the second

group, then, we include those cases of early cancer of the vocal cord which are either too far advanced or too unsatisfactorily located for operation by intralaryngeal dissection and which yet offer reasonable hope of cure without the necessity of total extirpation of the larynx.

The ideal case is one in which the lesion involves the middle portion of the vocal cord with both ends uninvolved. The operation of laryngofissure which, of course, consists of splitting the thyroid cartilage in the midline, thereby exposing its interior, is a brilliant technical conception, relatively simple to perform. As time will not permit a discussion of the steps in the procedure, only a few statements on some points of interest will be offered. We have in general adopted the standard type of operation, preferring not to resort to tracheotomy or excision of the thyroid ala. The operation is satisfactorily done under local anesthesia. An electric saw used too vigorously in cutting through an ossified thyroid may produce sufficient heat to destroy bone and result in sequestration. The thyroid cartilage in women may as a rule be divided in the median line by means of a knife. We have made it a routine procedure to use coagulation following the removal of the vocal cord. This, as a rule, controls postoperative bleeding. Some patients have developed granulation tissue which occasionally taxes our diagnostic ability as this tissue closely resembles a recurrence of the original lesion. Frequently it is necessary to remove this granulation tissue to provide adequate breathing space and to check up microscopically on the tissue removed. To date only twenty cases have been operated upon by this method. Seventeen patients have remained free from recurrence. They have been carefully selected and the results obtained have been entirely satisfactory. It has been necessary to reoperate on three patients with definite recurrences. Two of these responded to diathermy coagulation under suspension. The first is still well after four years; the second, shows no evidence of further recurrence after one year. The other patient reoperated upon has again developed a recurrence and will probably require a laryngectomy. One patient developed perichondritis following radiation. Three patients developed excessive scar tissue in the larynx producing signs of dyspnea. All three of these have required much attention because of the dyspnea.

Into the third group are placed those cases which, although considered too extensive for laryngofissure, are yet confined within the cartilaginous structures of the larynx. To the latter portion of this stipulation may be extended some latitude. Tumors that have extended slightly beyond the borders of the cartilaginous structures

or those exhibiting very early metastasis to the regional nodes may still be included in this division and operation by laryngectomy advised.

Lagging of the involved cord and certainly fixation of the cord are indications that the lesion is too far advanced for successful laryngofissure. Involvement of an entire cord or invasion across the anterior commissure with a considerable involvement of both cords is likewise a contraindication to laryngofissure.

Formerly ether or chloroform was thought to be the only satisfactory anesthetic for laryngectomy. While general anesthesia is still extensively employed for the operation, we have, for the past six years, used only local infiltration with novocain and are convinced of its superiority. We feel that reasonably heavy preoperative sedation, in spite of arguments against it, is safe and it certainly adds to the ease with which the patient can be made to cooperate. During this six-year interval it has been necessary to resort to a general anesthetic only once. This was in the case of a robust individual who, contrary to expectation, was made wildly excited by the preoperative sedative.

Ten years ago we discarded the old T-incision in favor of a simple midline division extending from just above the hyoid bone to the spurasternal notch. By making traction first on one side and then on the other, ample room has been furnished to carry out all of the necessary procedures. With the midline incision, closure is more easily accomplished and healing is far more rapid and uncomplicated.

The larynx is skeletonized by separately dividing the muscles at their attachment. The subperichondrial elevation as recommended by Crowe has been employed on several occasions, but in our experience it has not facilitated skeletonization. Next, the trachea is severed. If possible this is done just below the cricoid cartilage. In cutting through the trachea a small tongue-shaped flap is dissected from the posterior tracheal wall. This is thought to be a very important step for its proper utilization not only minimizes the possibility of drainage into the tracheobronchial tree, but it also produces a more generous mucosal area available for the formation of the tracheostomy.

As soon as the trachea is severed, it is lifted above the field to prevent the entrance of blood and a large rubber tube of approximately its same diameter is inserted into the lumen. Here the tube is firmly fixed by two or three large safety pins. The pins, held by

heavy forceps, are passed through-and-through to include both the trachea and the tube. This has a twofold purpose. It closes the trachea to the entrance of blood and it provides a means of traction upon the trachea which lifts it away from the larynx and elevates it from the level of any puddling of blood which could seep through an imperfect corking by the tube. Once this is accomplished, the opposite end of the tube is allowed to pass down the side of the table toward the head, where away from the scene of action and acting as an airway, it can be observed by the nurse seated at the head of the table.

The larynx is dissected from below upward and when it is finally extirpated, every effort should be made to preserve as much of the pharyngeal mucosa as possible. A piece of merthiolated gauze is then packed through the defect into the pharynx which has been previously slightly anesthetized and it is allowed to remain in place during most of the pharyngeal closure. When this is almost complete, the pack is removed through the mouth and the feeding tube is inserted, following which the remaining defect in the pharyngeal wall is closed. Meticulous attention to this place of the operation, we think, has materially lessened the incidence of fistula and secondary infection. At various times we have experimented with different suture materials, including black silk and cotton, for closure of the pharynx but we have come to the conclusion that No. 0000 chromic gut on atraumatic needles is superior to any other material. A layer of muscle is brought over the suture line and adequate drainage is obtained by making bilateral stab wounds slightly above the upper border of the trachea and about two inches laterally. A rubber dam is introduced through the stab wounds and placed appropriately within the operative field. These drains are left in place for five or six days.

At this stage, after all bleeding points have been secured, a generous amount of sulfathiazole powder is sprinkled into the wound. This applies only to the last six cases, as prior to them the drug was not in general use. We feel that healing has been more rapid and drainage lessened in these cases.

We have borrowed a procedure from the general surgeon which has been of considerable assistance. After applying a layer of gauze over the wound, a large sea sponge is placed over it and this is bandaged tightly, thereby obliterating any dead spaces by maintaining constant pressure. This type of pressure bandage is continued for about three days. By adhering to the above principles we have

had a high incidence of primary healing and the few esophageal fistulae have not been very troublesome.

Until the wound is completely healed, no attempt is made to encourage the development of a buccal voice. Some patients have developed excellent voices, one appearing on Ripley's program. Those who have not been successful in acquiring a pseudovoice have been urged to use an artificial larynx and some of these have developed speech with uncanny ability. Only one case has been regrettable. A successful merchant from a small town was advised to use a pad and pencil after his laryngectomy, but he could neither read nor write. He has learned to whisper after a fashion and remains well from an operative point of view, but he is morose and is a cause of grief in his family. Our observation is that the laryngectomized patients accept the problem of rehabilitation in a calm and determined manner, and in spite of this handicap they are a happy lot, thankful to be alive and anxious to do their bit in the world of today.

Of 48 patients upon whom laryngectomies were performed, 11 are living and well after five to 10 years; 23 are living and well after one to five years; six died within two years after operation; two died within six years after operation; and six patients have not answered questionnaires.

Into the fourth group are placed those cases which are too extensive for surgery, the so-called extrinsic type of carcinoma. Unfortunately too many such cases are seen for the first time by the laryngologist and usually little can be offered these patients. Radiation may prolong life but often enough the complications of radiation therapy only add to the misery already present. As far as cure is concerned, the hope is too forlorn to offer much in that direction. But let this not be considered an indictment of radiation therapy in carcinoma of the larynx in general. While we are convinced that surgery offers the greatest number of permanent cures in properly selected cases, the advocates of radiotherapy are providing sufficient evidence of success in similar circumstances to give their contentions careful consideration.

It is certain that if the general practitioner could more fully realize that life and death in carcinoma of the larynx depends upon an early diagnosis, a more conscientious effort would be made to arrange laryngological consultations for all persons suffering from hoarseness. It has always been an ordeal to inform the family and in occasional instances, the patient himself, that a growth is too

extensive for surgical removal. Each of you who has been through this knows the numerous questions with which the laryngologist is besieged and it usually ends with the statement that if only the dangers and potentialities of persistent hoarseness had been known, a physician would certainly have been consulted sooner.

Again, therefore, let it be well remembered that persistent hoarseness is the one and only unfailing indication of early carcinoma of the larynx.

OCHSNER CLINIC.

XLIV

EAR DROPS IN ACUTE OTITIS MEDIA*†

AN EVALUATION OF VARIOUS MEDICAMENTS AND AN ANALYSIS OF THE UNTOWARD EFFECTS OF ANTIPYRINE AND BENZOPYRINE

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AND

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For the relief of earache, heat as the sovereign remedy has come down through the ages. The methods most commonly employed were heated salt applied externally or warm water poured into the ear. In the days when bloodletting was a part of the physician's daily curriculum, some enthusiasts applied leeches to every painful ear.

When drugs and chemicals came into use, camphorated oil for the relief of eachache was accepted with favor, the sense of warmth imparted by camphor contributing to ease pain. Oil which has the property of retarding heat dissipation and is nonirritating to the drum formed the best vehicle for camphor. That this combination is logically sound is attested to by the fact that camphorated oil is still resorted to for earache by the laity, and this product is favorably looked upon by many of the profession.

However, phenol glycerin has been used by otologists all over the world for acute inflammation of the drum since the turn of the century. In the early days 10 to 20 per cent of carbolic glycerin was employed, but the concentrated solution was found to cause severe pain if perforation of the drum had occurred. From the instillation of the strong drops the membrana tympani occasionally assumed a macerated appearance. The 5 per cent concentration of carbolic acid in glycerin was unanimously accepted as the drops of choice for acute otitis media. Phenol is the best topical anesthetic

*Presented before the Otology Staff of Temple University Hospital, May 1, 1941.

†From the Department of Otology, Temple University School of Medicine.

for the cutaneous outer covering of the drum. This drug antiseptic izes the ear canal and blanches the hyperemic drum by constricting the blood vessels. Although glycerin is mildly irritating to an epidermal surface, its hydroscopic properties make this substance a most advantageous vehicle for drops for a congested drum membrane.

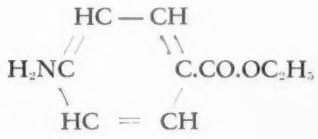
From time to time, solutions of the salts of silver, zinc and aluminum have been used as ear drops in acute otitis media. Some otologists have advocated irrigating the ear with an infusion of camomile in order to bring about resolution of the inflamed tympanum. For analgesia, a 10 per cent solution of opium in oil as ear drops has been a favorite preparation among the older practitioners. None of these solutions, however, gained much in popularity and most of them have been omitted in the newer textbooks on otology.

Bonain and Gray were the first to incorporate cocaine into mixtures for local anesthesia in surgery of the ear drum. According to Bonain's method, equal parts of cocaine, menthol and carbolic acid are rubbed together in a mortar until liquefied. A cotton-wound tip of an applicator is then dipped into the solution and applied carefully along the line of proposed incision. Gray's mixture consists of 15 per cent solution of cocaine in equal parts of rectified spirit and aniline oil. Fifteen to twenty drops of this preparation are instilled into the ear and allowed to remain there for about ten minutes. Just before the operation the anesthetic solution is wiped away. Gray[1] states that the danger of absorption of the cocaine is practically nil, but advises caution.

It appears logical to state that the capacity for the absorption of liquids by the intact ear drum is poor; that the epidermal layer of the membrana tympani is practically impervious to such substances as water, glycerin and mineral oils; and that as a general rule the aromatic vegetable oils are absorbed to a greater extent in keeping with the absorptive ability of the skin surface.

With the discovery that the anesthetic action of cocaine is due to its containing a benzoic acid radical in combination with a nitrogen basic group, synthetic chemists are now able to produce analagous compounds of lower solubility by reducing the basic nitrogen grouping.

Simple esters of aminobenzoic acid are slow in absorption. The instillation of a few drops of ethyl aminobenzoate in a 1 to 2 per cent solution in vegetable oil will afford considerable relief in a painful ear. However, even though the absorption is slow, the



above preparation cannot be recommended universally as suitable drops for earache because of its potential toxicity. Its usefulness should be limited to local anesthesia for surgery of the ear drum. An excellent solution for this purpose is one containing a concentration of 5 per cent phenol and 1 to 2 per cent ethyl aminobenzoate in vegetable oil.

Antipyrine as a local anesthetic has been given an extensive trial during the past fifty years. It has been widely used as a spray to relieve pain in pharyngitis and it is found incorporated in suppositories for painful hemorrhoids. It has also been injected hypodermically in the region of a hypersensitive nerve as a local anesthetic. However, in most branches of medicine, the employment of antipyrine as a local anesthetic agent has been abandoned. It was found to be a primary irritant, and cocaine has always been incorporated with it so that antipyrine might be tolerated. Moreover, the anesthetic powers of this drug are now known to be feeble. The same is true of its antiseptic properties. Goodman and Gilman[2] in their recent book describe antipyrine as being only mildly anesthetic and slightly antiseptic. Hare,[3] who was one of the greatest students of the coal tar products in his day, stressed the irritating qualities of antipyrine when applied locally. Of the untoward effects caused by this drug, he emphasized the fact that skin eruptions were by far the commonest manifestations. After medicinal doses of antipyrine, pemphigus-like spots appear occasionally, and often large bullae have been noted. We now include these manifestations under the term of drug allergy,[4] which may be caused either by ingestion, inhalation or local application of a medicinal agent. The untoward effects of antipyrine undoubtedly contributed to the dwindling of the popularity of this drug in medical practice.

The analgesic action of antipyrine benzoate (benzopyrine) is somewhat enhanced by its benzoic acid radical. In internal medicine, this drug has the same indications as antipyrine. When applied locally, however, occasionally it may cause contact dermatitis, an untoward effect ascribed to synthetic benzoates. Dentists' hands are particularly susceptible to procaine contact dermatitis and a similar effect from pontocaine has been described as affecting otolaryngologists.[5]

Strange as it may seem, great powers of antisepsis and analgesia have been ascribed to ear drops, the ingredients of which are 3.4 per cent of antipyrine, and 2.44 per cent of antipyrine benzoate in glycerin solvent. Some practitioners have been led to believe that resolution of the otitic infection is brought about by the instillation of these drops into the ear canal. It is, therefore, appropriate to point out the fact that the conception of modern otology is that the mastoid cells become involved in the infectious process at the same time as the drum and middle ear. How, then, could gr. 1/6 of antipyrine, when deposited in the external auditory canal, sterilize the temporal bone plus the eustachian tube and nasopharynx? Pain cannot be relieved unless 1) the serum or pus contained in the middle ear may find an escape through the eustachian tube when the congestion about the orifice of the canal subsides, or 2) drainage be provided by a perforation in the membrana tympani.

A great deal of unnecessary trouble has been caused by the injudicious use of a proprietary preparation consisting of antipyrine and benzopyrine in a glycerin solvent that is prescribed empirically and is also sold indiscriminately over the counter for earache.

It requires no lengthy discussion to substantiate the statement that this drug which is generally viewed as an antiseptic or an analgesic is at times more harmful than good.

We can cite an experiment where 10 normal ears were treated with it for twenty-four to forty-eight hours to determine definitely its action upon normal tissue. Four of these cases terminated with bullae which were followed by pain and which did not subside until the blebs either ruptured spontaneously or were ruptured mechanically; the remaining cases (6) showed no ill effects from this drug. It would seem, then, that with the incidence of 4 out of 10 normal cases showing some ill effects from this drug, this proprietary agent should be prescribed with more judiciousness than has been practiced.

We have also seen the development of blebs, single, multiple or hemorrhagic, in at least 35 per cent of our patients after the use of this preparation. This may be ascribed either to the irritating effect of the mixture, contact dermatitis from the chemical, or drug allergy.

When the bleb forms as a result of the drug, there is a recurrence of pain and frequently a rise in temperature. The pain does not subside until the bleb ruptures spontaneously or is broken

mechanically, either by incision or by rupture with an applicator. These blebs give the appearance of a bulging ear drum and are often difficult to differentiate from acute suppurative otitis media, otitis media bullosa or the drug bleb. This condition when it arises, calls for the experienced otologist to exercise his judgment as to procedure.

We know that in a true case of suppurative otitis media, myringotomy is indicated. However, in cases merely of drug bullae, whether they be hemorrhagic or otherwise, the technic of simple evacuation should be followed without incision of the ear drum. In cases of otitis external bullosa which are often found in influenzal infections, the blebs are emptied without incision of the ear drum. It is vital, therefore, with the high incidence of bleb formation due to the indiscriminate use of these so-called "ear drops" that the physician make certain that he is merely dealing with a drug bleb instead of an otitis media, as unnecessary myringotomies often result in middle ear infection. Furthermore, as this confusion does occur and gives rise to uncalled for complications, such untoward effect should constitute a potent argument for caution in the routine use of antipyrine and benzopyrine in glycerin as ear drops.

SUMMARY

1. Various medicaments employed as ear drops have been analyzed.

2. The instillation of antipyrine-antipyrine benzoate-glycerin solution into the external auditory canal appears to be contraindicated.

1915 Spruce St.

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XLV

THE ETIOLOGY OF MYRINGITIS BULLOSA HEMORRHAGICA*†

Preliminary Report

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Although the existence of myringitis bullosa hemorrhagica has been recognized by authorities for many years, there has been little unanimity of opinion as to its actual cause. No careful etiological studies have appeared in the literature. In 1902, however, Politzer,[1] in his comprehensive description of inflammatory changes of the tympanic membrane, indicated that hemorrhagic blisters are frequently seen in the otitis due to influenza. During the great influenza pandemic of 1918-1919 numerous authorities associated bullous hemorrhagic lesions of the ear drum with influenzal infection. As a result, these lesions were referred to as "influenzal blebs" by otologists, and their mere presence accepted as pathognomonic of epidemic influenza.

In a careful survey of almost 7,000 cases of influenza during the first World War, Hill[2] observed only 120 cases with involvement of the middle ear. This report is a carefully conducted observation of the pathologic changes occurring in the more severe form of myringitis bullosa hemorrhagica. As the disease progressed Hill observed an actual hyperplasia of the mucous membrane of the middle ear with an obliteration of all the normal landmarks of the tympanic membrane.

Bryan and Howard[3] reported 1,500 cases of influenza which occurred during the pandemic of 1918, only 37 of which showed otitic complications. At Camp Sherman, where 11,000 cases of in-

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†This work was supported by the Rose Lampert Graff Foundation, Los Angeles, California.

fluenza occurred, Westlake[4] noted that myringitis bullosa hemorrhagica was encountered in relatively few cases.

No further reference is made to this condition until a short clinical report by Ziegelman[5] in 1931. This investigator theorized that circulating toxins with an affinity for the tympanic membrane were the cause of "influenzal blebs". In a study of 87 cases of myringitis bullosa hemorrhagica occurring in children, Karelitz[6] observed that more than one-half complicated "acute infection of the respiratory tract." More recently, Ruskin,[7] in the light of the earlier work of Hess[8] on infantile scurvy, suggested the possible relationship between a scorbutic state and the occurrence of hemorrhagic blebs on the tympanic membrane.

In view of the divergent theories of previous workers it seemed advisable to investigate the true etiology of myringitis bullosa hemorrhagica. During the past two years a large series of these so-called "influenzal blebs" has been observed. Over 100 cases were seen in clinic and private practice during the winter months of 1940-1941. Simultaneously a nation-wide outbreak of epidemic influenza[9] occurred, suggesting a possible relationship between these two conditions.

Six cases of myringitis bullosa hemorrhagica complicating upper respiratory infection in adults were selected for study. The blebs varied in size from small hemorrhagic vesicles to purplish grey, crinkled membranes which completely obscured the drum and extended over the annulus onto the posterior-superior wall of the external auditory canal. Frequently several distinct blebs were visible, confined to the posterior quadrants and giving the impression of a sagging posterior canal wall. Careful early evacuation of these blebs or spontaneous external rupture revealed a raw ulceration of the superficial epithelium, leaving the remaining membrane intact. As the inflammatory process subsided, the drum was frequently covered by a purplish black, shriveled membrane which was removed with difficulty, thereby preventing satisfactory observation of the healing process.

Two of the cases under investigation showed bilateral otitic involvement and four were unilateral; the blebs varied in size and number. Spontaneous rupture of the blebs occurred in four ears. Involvement of the middle ear occurred in three cases, in one of which there developed a serous otitis media which subsided under inflation of the eustachian tube, the other two requiring myringotomy. Typical sagging of the posterior-superior canal wall with subsequent spontaneous healing occurred in two cases.

Because it is the purpose of this study to determine whether a relationship exists between myringitis bullosa hemorrhagica and epidemic influenza, a brief discussion of the etiology of the latter disease is pertinent. Since 1918 a number of different agents have been described as the cause of influenza. It was not until 1933, however, that Smith, Andrews and Laidlaw[10] succeeded in isolating a filtrable virus which proved to be the true cause of the disease. This virus became known as "influenza A". These workers also demonstrated that antibodies against the virus were produced during convalescence. Stuart-Harris, Smith and Andrews,[11] as well as Rickard, Lennette and Horsfall,[12] later demonstrated that only a certain proportion of the cases studied in a given outbreak were due to influenza A virus and suggested that epidemic influenza was not a single etiological entity, but that more than one agent was capable of producing the disease. Recently Francis[13] recovered a virus different from influenza A and demonstrated its causal relationship to the disease. This agent has been termed influenza B virus and it is now agreed that influenza A and influenza B are etiologically distinct although clinically similar diseases. A third type of clinical influenza, now referred to as influenza X or Y, is an acute febrile disease of unknown etiology in which neither the A nor the B virus can be recovered and in which antibodies specific for these viruses cannot be demonstrated during convalescence.

Certain laboratory tests (complement fixation and neutralization tests) now available for the study of epidemic influenza offer a convenient method for making a specific diagnosis. In the case of either test, however, sufficient time must elapse between the onset of the disease and the date on which the convalescent serum is obtained for the production of additional antibodies by patients infected with the virus.

Accordingly, in our series when the ear was first examined and the diagnosis of myringitis bullosa hemorrhagica made, an acute-phase serum specimen was obtained from each patient. Fourteen to eighteen days later a convalescent-phase serum specimen was taken. The technic of the neutralization and complement fixation tests used to determine whether the patients had influenza, was as follows:

The PR-8 strain of epidemic influenza virus* isolated by Francis[14] was used in the neutralization tests. Stock suspensions (20 per cent by weight) of infected mouse lungs, prepared by Horsfall,[15] were used as the source of virus. Albino

*The authors are grateful to Dr. George K. Hirst of the Rockefeller Foundation for the PR-8 strain of influenza virus.

Swiss mice between three and four weeks of age were used in the test. The sera were inactivated by heating to 56° C. for 30 minutes. The acute and convalescent serums from a given patient were treated at the same time against one suspension of the PR-8 strain of epidemic influenza virus. Serial fourfold dilutions of serum were made in 0.85 per cent NaCl, the volume of the final solutions being 2 c.c. Serial tenfold dilutions of the stock 20 per cent suspension of infected mouse lung were made in beef infusion broth containing 20 per cent normal horse serum, and in the test a 10^{-3} dilution representing approximately 3,000 of the 50 per cent lethal doses of virus was used. The desired serum dilution (0.3) was thoroughly mixed with 0.3 c.c. of the appropriate virus dilution and the mixtures were incubated at room temperature for 30 minutes. In testing the acute-phase serum, four mice were used in a group receiving a particular serum dilution and virus mixture, while six mice were used in a group when testing the convalescent-phase serum. Each mouse was inoculated intranasally under light ether anesthesia with 0.05 c.c. of respecive serum-virus mixtures. The animals were observed daily.

Mice which survived the observation period of 10 days were sacrificed and their lungs examined for the presence of pulmonary consolidation. The 50 per cent end-point method of Reed and Muench[16] was used to determine the highest dilution of serum which was capable of neutralizing a given dilution of the virus suspension.

The procedure of the complement fixation tests was similar to that described in a previous communication.[17] Both acute- and convalescent-phase serum specimens were tested at the same time. The supernates of a 10 per cent suspension of lung from Swiss mice infected with the type A (PR-8) and type B (Lee*) strains of influenza virus were used as stock antigens. The antigens were titrated for hemolytic, anticomplementary and antigenic units, and it was found that 0.5 c.c. of a 1:10 dilution of the stock antigen was suitable for the test.

On the basis of preliminary anticomplementary titrations of the antigens three minimal hemolytic doses of complement were found adequate for the test. Fixation was accomplished by incubation for one hour at 37.5° C. followed by 18 hours at 4° C. After the addition of the hemolytic indicator system, the test was reincubated for one-half hour in the 37.5° C. water bath.

Complement fixation tests were done on the acute- and convalescent-phase serum specimens from each of the six cases of myringitis bullosa hemorrhagica, using mouse lung suspensions of type A and type B virus as the respective antigens. Two known cases of type A influenza and one case of type B influenza were included as controls. Neutralization tests were performed to confirm the specificity of the complement fixation test. Serum from each of three cases of myringitis bullosa hemorrhagica and two cases of type A influenza (controls) were tested by this procedure. The type A strain (PR-8) of virus was used in these tests.

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*The Lee strain of type B influenza virus was kindly supplied by Dr. Thomas Francis, Jr., of the University of Michigan School of Public Health.

TABLE I

Results of Neutralization and Complement Fixation Tests With Epidemic Influenza Virus on Acute and Convalescent Phase Blood Specimens From Each of Six Patients With Myringitis Bullosa Hemorrhagica

Patient	Serum	Neutralization Tests*	Complement-Fixation Test Type A†	Type B‡
1	Acute	.....	1:8	0
	Convalescent	.....	1:8	0
2	Acute	.....	0	1:8
	Convalescent	.....	0	1:16
3	Acute	.....	1:8	1:8
	Convalescent	.....	0	1:8
4	Acute	1:10	1:16	0
	Convalescent	1:5	1:16	0
5	Acute	0	0	0
	Convalescent	0	0	1:8
6	Acute	1:6	1:8	0
	Convalescent	1:9	1:8	0
7	Acute	1:6	0	1:16
(Control)	Convalescent	1:90	1:64	1:8
8	Acute	1:10	1:8	0
(Control)	Convalescent	1:150	1:64	0
9	Acute	.....	1:8	0
(Control)	Convalescent	.....	1:8	1:32

*Serum titers expressed in terms of neutralizing capacity against approximately 3,000 of the 50 per cent mortality doses of type A virus (PR-8 strain).

†Infected mouse lung suspension of type A virus (PR-8 strain) used as antigen.

‡Infected mouse lung suspension of type B virus (Lee strain) used as antigen.

The results summarized in Table I indicate that none of the patients with myringitis bullosa hemorrhagica showed evidence of infection with influenza A or B viruses. No increase in specific complement fixation or neutralizing antibodies against these viruses could be demonstrated in the convalescent-phase serum specimens from these patients. The control cases of influenza A and B, however, showed increase in specific antibodies in their respective convalescent-phase sera. It is impossible at the present time to ascertain the relationship of myringitis bullosa hemorrhagica to the third type of clinical influenza now referred to as influenza X or Y, since its causative agent has not as yet been isolated.

Although attempts to demonstrate other filtrable agents in the fluid aspirated from these bullous hemorrhagic lesions of the ear drum have thus far been negative, experiments of this nature are still in progress.

CONCLUSIONS

1. The results of this study indicate that no relationship exists between myringitis bullosa hemorrhagica and the known types of epidemic influenza.

2. The term "influenzal bleb" as frequently applied to these bullous hemorrhagic lesions of the ear drum is misleading.

3. The etiology of myringitis bullosa hemorrhagica is still under investigation.

FRISCO BLDG.

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XLVI

BROKEN NEEDLE FOREIGN BODY IN THE TONSILLAR FOSSA*

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The harrowing experience of losing a segment of a broken needle in the pharynx during the course of a tonsillectomy has occurred in the experience of a number of laryngologists. This may be substantiated by anyone recalling random conversations in which such cases have been mentioned "off the record". Considering that tonsillectomy is the operation most frequently performed in this country—in the majority of cases by men outside of the specialty—it is a reasonable presumption that the unfortunate accident of breaking a needle is not uncommon. It is regrettable that this troublesome occurrence has not been discussed more freely and frankly for the guidance and instruction of colleagues encountering the dilemma.

The almost total absence of such reports in the literature may be due to the fact that no individual has had sufficient experience with broken needles to warrant an article on the subject. For this reason the questionnaire method was employed in order to correlate the experiences of a group of representative laryngologists whose contact with the mishap of a broken needle may have occurred in practice, consultation or the training of interns.

The purpose of this paper is to call attention to the heretofore unreported relative frequency of breaking a needle during tonsillectomy; to present the results of a questionnaire survey of cases in respect to causes, symptoms, management, complications and sequellae; and to indicate means of avoiding the accident. A simple efficient method of removing a needle foreign body from the tonsillar fossa will be described. Finally, the medicolegal aspect of the situation will be discussed briefly.

A complete discussion of various methods employed in the removal of tonsils is not germane to this report. The individual laryngologist is privileged to use the procedure that he has found

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most efficient and trustworthy. To secure actively bleeding vessels, most surgeons resort to either ligatures or sutures. Ligatures are placed by tying over a hemostat clamping the bleeding vessel or with the aid of a variety of tiers or ligature-tying forceps, such as the Boettcher, Howard, McWhorter, or similar instruments. Other operators have greater confidence in transfixing the bleeding area by various types of suture needles. The Hourn needle and its several modifications (Dupuy, Whelan, Hinton, Goodspeil), consisting of a handle with the distal end formed into a needle, are in general use. If this type of needle is broken, the fragment is still held by the suture material and cannot be lost. Automatic suture needles of the Shuster type have some use. A number of surgeons, however, prefer to use a favorite needle-holder with a separate needle suitable to the location of the bleeder and the size of the fossa. With this technic the breaking of a needle and loss of a fragment may occur. This accident does not condemn the method entirely but stimulates inquiry into the factors relevant to the situation.

A suture is probably more reliable than a ligature in preventing postoperative bleeding and it can be placed more securely in all parts of the tonsillar fossa. Hemostasis effected by suturing freely bleeding vessels is definitely conducive to a tranquil surgical conscience.

Since suturing with a separate needle held in a suitable needle-holder is in general use and is an accepted method of controlling bleeding after tonsillectomy, it is advisable to consider the reasons for the breaking of a needle and, of necessity, the management of the accident when it occurs. The problem is not limited to broken suture needles, but includes, as well, needles used for the injection of a local anesthetic for tonsillectomy.

The following reasons may account for breaking a needle: (1) defective needle, poorly tempered, etc; (2) the weakening of a needle due to previous bending; (3) movement of the patient's head when the needle is engaged in the tissues; (4) unsuspected solid structure in the tonsil, as an elongated styloid process or a cartilaginous or bony tonsillith; (5) excessive stress on the needle due to inept handling of the needle-holder.

The majority of accidents may be charged to the operator and could be avoided by proper dexterity and care. The above causes, except the fourth, apply as well to the breaking of the usual type of needle used in local anesthesia, namely the "tonsil needle". The break always occurs at the junction of the rigid

tubular shank and the thinner needle point brazed to the distal end.

The operator must be alert to the various possibilities after the breaking of a suture needle in the tonsillar fossa in a patient under general anesthesia. The lost segment may be: (1) partially imbedded in the fossa; (2) completely buried beneath the surface of the fossa, i.e., beneath the superior constrictor muscle and therefore in the parapharyngeal space; (3) in the larynx or tracheobronchial tree; (4) in the pharynx or nasopharynx as a free foreign body; (5) in the tubing or bottle of the suction apparatus; (6) in the stomach.

The patient's head should be lowered at once (if it is not already in the Rose position). The pharynx and fossa should be cleared by aspiration. Persistence of bleeding handicaps the surgeon in his search; vomiting adds to the difficulties. If the needle fragment is not seen at once, the bleeding must be controlled before further search is carried out. The next step is the careful inspection of the nasopharynx and larynx. The Flagg battery-handle type of laryngoscope may be used for viewing the piriform fossa, vallecula, larynx and upper trachea. The contents of the suction tubing and bottle should be examined.

If these steps are unsuccessful, the patient must be transported to the X-ray department while still under anesthesia. Fluoroscopic examination may show the needle in the tracheobronchial tree, necessitating prompt bronchoscopic removal. If the needle has been swallowed, it may be observed in the stomach. A needle imbedded in the fossa is easily visualized as a rule. Roentgenograms should always be taken for permanent record. Even if the needle is not located by fluoroscopy, complete roentgenograms must be taken from the head to the diaphragm, including lateral and anteroposterior views of the neck.

The presence of the needle buried in the fossa requires an immediate attempt at removal while the patient is still under anesthesia, if the general condition does not contraindicate. The necessary instruments, including a suction machine, should be set up in the X-ray department. Using the Flagg laryngoscope for depressing the tongue as well as for illumination, a slender-jawed artery forceps is brought into contact with the surface of the fossa. The laryngoscope is then withdrawn and the handscreen type of operating roentgenoscope is used. Under fluoroscopic guidance the exploring forceps is maneuvered until the tip overlies the needle.

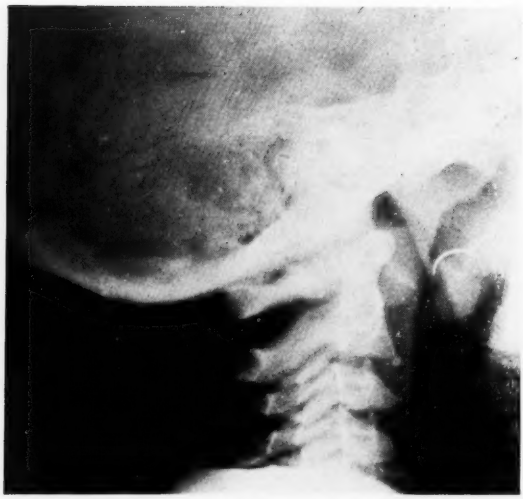

Fig. 1.—Case 1. Broken needle segment imbedded below the surface of the tonsillar fossa.

The instrument is then gently inserted into the tissues until the tip reaches the depth of the foreign body. When contact has been established the jaws of the forceps are opened and the needle is grasped. It may then be twisted until one end is brought into view and seized with another forceps. As an alternative, after the needle has been fixed with the forceps, dissection may be done under direct vision until it is exposed.

The handscreen facilitates precise localization and guides the searching instrument, thus avoiding extensive traumatic dissection. It is small enough to permit freedom of action by the operator. Fluoroscopic guidance is indispensable in the technic.

The extraction of a fragment of a needle used for the injection of a local anesthetic is not simple even if the point of penetration is apparently known. A preliminary roentgenogram should be taken at once. The tonsil is then removed, preferably with the patient in the horizontal position to prevent aspiration if the needle is dislodged by dissection in the capsular space. If the needle is not discovered, further search should be undertaken under fluoroscopic observation.

If a needle has been left in situ, periodic inspection and palpation of the fossa are advisable to discover spontaneous partial extrusion. Also, roentgenograms may be taken at intervals to observe possible migration.

One must consider the hazard of ether explosion if the fluoroscopic apparatus is not sparkproof. In delayed attempts to recover a needle other anesthetics may be useful, such as intravenous pentothal or nitrous oxide by intratracheal insufflation.

Of the 180 questionnaires sent out, 65 (36 per cent) were returned. Of these 34 reported broken-needle accidents (52.3 per cent of the returns, or 18.8 per cent of the number of inquiries). The 34 men answering in the affirmative reported a total of 67 broken needles. Of these, 10 were local anesthesia needles, though this question had not been included in the questionnaire, and 57 were suture needles. The number reported by any individual ranged from one to six cases. The needle was removed in approximately 72 per cent of the cases (48 of the 67).

Known complications or sequellae occurred in eight cases (12 percent): (1) fracture of the hyoid bone during the attempted removal of a needle, with subsequent extrusion of a sequestrum; (2) persistent neuralgic pain distributed over the course of the glossopharyngeal nerve; (3) two cases of parapharyngeal space infections; (4) four cases with variable degrees of pain on swallowing.

In a majority of the cases the patient was unaware of the presence of a retained needle. There were no reports of excessive hemorrhage or pulmonary suppuration. In two cases subsequent roentgenograms showed migration of the needle.

CASE REPORTS

The following case reports are representative of most of the situations that may be encountered. The first was the author's case; cases 2, 3, and 4 were those operated upon by members of the house staff under his supervision. Cases 5 to 12 were obtained from the questionnaire. Case 13 was the only report found in the literature.

CASE 1.—In attempting to secure a bleeding tonsillar vessel high in the vault of the fossa in a 3-year-old child, the suture needle broke and the loose segment remained completely imbedded under the surface. Roentgenologic examination showed the needle in the tissues (Fig. 1). Using the method described above, the missing needle was located and removed in about six minutes. There was no subsequent bleeding and recovery was uneventful.[1]

CASE 2.—In the course of a tonsillectomy on a child of 6 years of age under general anesthesia, a suture needle was broken, leaving one end projecting above the level of the fossa. This was easily removed with a hemostat.

CASE 3.—During a tonsillectomy on a child a medium-sized suture needle partially inserted into the tissues slipped from the needle-holder. At the same time the catgut was pulled out leaving the needle as a free foreign body. After aspiration of the pharynx, a careful search revealed the needle in the nasopharynx.

CASE 4.—While injecting between the posterior pillar and the base of the tonsil for a local tonsillectomy, the operator broke the distal tip of the "tonsil anesthesia" needle. The fragment could not be seen. Fluoroscopy revealed the needle to be parallel to the mandible. The operation was completed. Roentgenograms taken immediately thereafter failed to show the needle anywhere in the neck or lungs. It is probable that it had become dislodged by gagging and was either swallowed or expectorated.

CASE 5.—While sewing the pillars together at the upper pole, the surgeon broke the suture needle leaving the free fragment buried in the tissues. Roentgenograms showed the needle fixed in the fossa. On the second postoperative day a futile attempt was made to remove the needle using a giant magnet. Four weeks later, Dr. S. J. Pearlman, in consultation, successfully removed the needle by dissection through the anterior pillar using the hand roentgenoscopic screen for guidance as in the author's first report.

CASE 6.—A patient with a needle which had been in the fossa for six weeks was seen in consultation by Dr. M. Cottle. Under the guidance of a head fluoroscopic screen in the form of a viewing box fitting over the surgeon's eyes, an artery forceps was snapped in the fossa overlying the needle. Then, under direct vision, dissection in the established location was successful in recovering the needle in fifteen minutes.

CASE 7.—A needle segment had been lost in the tonsillar fossa of an adult after a local tonsillectomy. One attempt at removal ended in failure after thirty minutes of exploration. Dr. D. A. Willis, in consultation, removed the needle using a head fluoroscopic screen and his "electrical forceps" which flashes a light when complete contact is made with a metallic foreign body.

CASE 8.—A needle was lost by an assistant in the course of a tonsillectomy on a 9-year-old boy. Roentgenograms showed the needle but proved to be an inadequate guide for removal by dissection. Dr. F. W. Davison then inserted a skin clip into the fossa. A new roentgenogram showed that the needle corresponded to the clip in the coronal and lateral planes, but was slightly higher in position. The following day, under local anesthesia, a transverse incision was made into the soft palate above the tonsillar fossa down to the point of localization in the roentgenogram. A metal bar in contact with a magnet was inserted into the incision and removed the needle.

CASE 9.—In a patient seen in consultation by Dr. J. B. Fred three months after the accident, a needle was successfully removed with the aid of a regulation fluoroscopic screen. The biplane fluoroscope was of no assistance.

CASE 10.—A patient seen in consultation by Dr. Noah Fox eighteen months after a broken needle had been left in place had neuralgic pain which was glossopharyngeal in distribution.

CASE 11.—Removal of a needle without utilizing a fluoroscope required two attempts and over two hours of operating time.

CASE 12.—In a patient seen in consultation by Dr. J. Theobald, an external approach to the parapharyngeal space was carried out to remove a needle foreign body on the day following the tonsillectomy.

CASE 13.—While the surgeon was suturing in the tonsillar fossa after a local tonsillectomy in a made adult, the needle point struck something hard and snapped in two. A roentgenogram showed the missing needle fragment and also the cause of the accident—an elongated styloid process. A two-hour search was unsuccessful. Six months later the styloid was exposed through the tonsillar fossa, its end amputated and the needle recovered.[2]

DISCUSSION

Various procedures have been employed in the management of the troublesome problem of needle foreign body. The method of "blind" dissection, either via the anterior pillar or through the fossa, usually involves a protracted operation if unaided by fluoroscopic control. Successful removal has not always followed tedious search even on repeated trials.

The giant magnet has been useless for removal of an imbedded needle fragment. Davison, however, used it after dissection had exposed the needle. LeJeune states that it has given some aid in localization by causing noticeable traction on the tissue above a buried needle.

The biplane fluoroscope, if available, could possibly be utilized to advantage. Fred, however, did not find it of particular assistance (Case 9).

The head type of fluoroscopic screen worn over the surgeon's eyes has been of value. The room remains light for the assistants while the operator uses the screen for guidance (Case 6).

Willis' method, successfully used in Case 7, depends upon two factors: first, fluoroscopic visualization and, second, the use of an ingenious forceps wired so that closure of the jaws on a metallic object completes an electric circuit and lights a battery-controlled signal lamp. The forceps serves both to locate and to grasp the object.[3]

In many instances of losing a needle the operator has left it in situ, making no attempt at recovery and, incidentally, often avoiding informing the patient. The procedure involves a gamble on the part of the surgeon: first, that no sequellae or complications will result; and, second, the certain risk of implied negligence and

guilt if the foreign body is discovered later. Needles have been retained without causing symptoms. Nevertheless complications do occur frequently enough to warrant removal of a lost needle. Also, the possible mental hazard of anxiety of the patient in the case of a retained needle should not be disregarded.

The problem of the broken needle may be eliminated by substituting other methods of control of bleeding after tonsillectomy. The questionnaire revealed that a number of men were strongly opposed to suturing after tonsillectomy, an attitude which is valid and to be respected. Nevertheless, since suturing is still preferred by some operators, the selection of the method of hemostasis must be left open to individual choice. Consequently a discussion of the means of prevention of accidents and their management is still pertinent. The problem of broken anesthesia needles remains even if needles are not used for suturing.

The incidence of breaking a needle may be decreased by (1) using needles of good quality, (2) discarding bent or damaged needles, and (3) exercising care and dexterity in the use of the needle-holder and the anesthesia syringe.

While our professional concern is primarily the welfare of the patient, a consideration of the medicolegal aspects of foreign-body accident also must be borne in mind.[4]

The breaking of a needle under most ordinary circumstances is considered a simple accident beyond the control of the surgeon. Malpractice is based upon "the formula of the four 'D's' ": (1) duty, the implied obligation when a physician undertakes the care of a patient, charity or private; (2) dereliction, failure to use average knowledge, skill, care and judgment (a specialist professing a high degree of competence is judged by the average practice of fellow-specialists); (3) Damage, to the patient as a result of the above; and (4) direct causation, direct relationship between dereliction and damage. If all of these are not proved, malpractice cannot be established.

The surgeon must assume certain responsibilities. He should exercise a number of precautions to avoid the unjustified stigma of gross carelessness and implications of guilt. These include: (1) informing the patient, or, if this is deemed inadvisable, a responsible relative; (2) notation of the accident on the hospital record signed by the surgeon; (3) notification of the hospital authorities; (4) prompt taking of an adequate number of roentgenograms to indicate the presence or absence of the foreign body; and (5) careful,

skillful procedure to recover the foreign body. The foregoing steps will indicate that no concealment of the accident or evasion of possible responsibility is intended.

SUMMARY

1. The breaking of a suture needle or an anesthesia needle in the tonsillar fossa is an unfortunate accident that is more common than has been stated.

2. Four cases are presented. In three instances the needle was successfully removed; in the fourth it was evidently swallowed or expectorated. Nine collected cases are quoted.

3. A questionnaire survey revealed reports of 67 additional cases of broken needle foreign body. Of these, 48 (71.6 per cent) were removed and 19 (28.4 per cent) remained in place. There were eight complications or sequellae (12 per cent) as follows: one case of fracture and extrusion of a part of the hyoid bone, one case of neuralgia of a glossopharyngeal nerve type, two cases of parapharyngeal space infection and four cases of dysphagia.

4. The removal of a needle foreign body should be attempted at the original operation.

5. The procedure to be followed is outlined with emphasis on the necessity of fluoroscopic visualization and control. For this purpose a small handmirror type of screen has been found of practical value.

6. Necessary precautions to avoid the accident are submitted.

7. The probable causes of the accident are mentioned.

8. The medicolegal aspect is considered.

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XLVII

ABSCESS OF THE LARYNX*

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This condition is of interest because of its infrequency and its high mortality rate. The first case of abscess of the larynx was reported by Roland in the thirteenth century. Laënnec reported two cases. Morrell McKenzie reports thirteen cases in his textbook. A. Mayer, who published a comprehensive paper in 1931 on abscess of the larynx, found only 30 such cases occurring in a period of eighteen years among a total of 73,413 patients examined.

Abscess of the larynx formerly occurred more frequently as a complication of typhoid fever. In 365 typhoidal abscesses of the larynx, more than one-half died. Of the 365, there were 243 tracheotomies, with 63 per cent recoveries. Of the remaining 122 treated without tracheotomy, only 14 recovered. More recently, abscess of the larynx is becoming common as a complication following in-dwelling feeding tubes and Wangensteen continuous suction drainage.

The following terms are descriptive of various stages of its development and may be used in describing the condition, depending upon the stage reached: (1) edema of the larynx, (2) perichondritis of the larynx (3) abscess of the larynx, (3) phlegmon of the larynx.

In infants, abscess of the larynx is a rare occurrence. In fact, Ballin quotes several authorities to support his statement that it is never found in children. However, Descottes in 1912 summarized the reports of 30 cases of abscess in children ranging in age from 5 weeks to 9 years of whom 11 were 2 years old or less. It is my personal opinion that clinical diagnosis of enlarged thymus is too frequently mistaken for this condition in children. Since opportunities for post-mortem examination are so limited in young children, description of the pathologic condition therefore leaves much to be desired. Errors in diagnosis are therefore the result. About 60 per cent of cases of abscess of the larynx (Woodson) occur in

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

individuals between the ages of 25 and 40 years. It is more common in the male than in the female in the ratio of 3:1. It is still uncertain whether or not the involvement of the larynx takes place via the blood stream in the cases of typhoid fever. It is the opinion of Chevalier Jackson and others that pressure necrosis and ulceration develop as a result of a prolonged flat position of the body that causes the larynx to press back onto the pharyngeal wall, leading to an infection of the laryngeal structure. Undoubtedly, the same mechanics intervene and produce the involvement in those cases caused by in-dwelling feeding tubes (Levin and Wangensteen). In addition to typhoid fever, scarlet fever, measles, typhus, septicemia, puerperal sepsis, erysipelas and syphilis, can be considered as possible causes of abscess of the larynx. Herpes zoster is a cause; metastatic infection undoubtedly can be a source. Traumatism, including that produced by intubation and endoscopy, certainly must be considered. Blows on the front of the neck by baseballs and bicycle handlebars are frequent causes. Acute tonsillitis and acute infection of the islands of lymphatic tissue in the vicinity of the larynx can be the causes in certain cases.

Pain may or may not be present, or it may be present only on pressure. By grasping the Adam's apple between the thumb and forefinger pain may be elicited. Hoarseness is invariably present, but it may not be marked. Dyspnea is often present but may not be recognized until the patient has developed a most dangerous condition. Occasionally, the epiglottis is involved alone. However, this is easily recognized and subsides rapidly on evacuating the pus. When the arytenoids are involved, they are seldom alone affected. Perichondritis of the thyroid cartilage can be easily diagnosed if there is tender swelling over the cartilage. Infection starting within may break through and appear externally. This is particularly true in small children. This stage can be confused with cold abscesses of tubercular origin and with infected thyroglossal cysts. In these cases abscess of the thyroid cartilage can burrow downward, without showing many signs, and lead to mediastinitis. In infections of the thyroid cartilage, because the upper and posterior portions of the thyroid cartilage form an unyielding wall externally, the floor and outer wall of the pyriform fossa bulge upward. Bulging, here, is a pathognomonic sign of perichondritis or abscess of the thyroid cartilage. If the floor alone bulges, the swelling may be coming from both cricoid and thyroid exudation. The vocal cords are usually not seen because of the swollen ventricular bands. The arytenoids are usually covered, or almost covered, by edematous tissue and are naturally made immobile by the swelling. Marked

swelling of the ventricular bands means involvement of the thyroid cartilage, whereas swelling of the laryngeal aperture indicates more involvement of the cricoid cartilage. The infection spreads either above or beneath the perichondrium, and necrosis and sequestration of the cartilage occur by reason of the interference with the nourishment to the cartilage. The perichondrium may remain intact, with pus burrowing between it and the cartilage, very much the same way as abscess of the nasal septum. We all know that only after long-continued septal abscess does the cartilage become necrotic, but, if free drainage is established, necrosis of the septal cartilage will be prevented. The ossified part of the cricoid is apt to necrose earlier; therefore, since ossification is well underway in the third decade, osteomyelitis is more apt to accompany infections of this kind in the adult, while perichondritis is apt to be alone the condition in childhood. The diagnosis can be easily missed because, oftentimes, the laryngeal involvement may appear much less in extent than it really is. Sudden death may occur, and may not be due entirely to asphyxia. Cardiac failure of toxic origin or sudden paralysis of the respiratory center may be the cause. Direct laryngoscopy is necessary to make a positive diagnosis in children. Indirect examination by throat mirror may suffice in adults. Dyspnea in any young child is an urgent reason for direct laryngoscopy, which is the only means by which precise diagnosis and treatment may be accomplished.

Unfortunately, there are two schools of thought regarding treatment that vary widely. There are those who advocate severely conservative treatment, and there are those who advocate most radical surgical procedures. They all agree, however, that early tracheotomy is necessary where dyspnea is even a mild symptom. Chevalier Jackson, Sr., has told me personally: "Any incision into the cartilage is to be gone into only after long waiting, and then only when definite pointing up begins to show that pus is present. Too free cutting into cartilage has a tendency to spread infection to new areas of cartilage. Since any destroyed cartilage is replaced by fibrous tissue, lumen of the trachea is bound to be narrowed. Therefore waiting for nature to sequestrate is apt to lead to less destruction than too early and too free incisions. Utmost conservatism is called for in treatment of these conditions. The ordinary principles applying to cartilages elsewhere do not apply to laryngeal cartilages in a state of inflammation of the perichondrium. It is better for necrotic sequestra, if any, to work their way out. Cutting down on the cartilage, scraping, and similar procedures, usually result in total obliteration of the larynx."

Quoting from Schenck, who believes in more radical procedure: "Treatment has been based largely upon experience gained in the typhoidal abscesses of the larynx. Drainage by incision is adequate in many of the milder cases; but laryngotomy must be resorted to in the treatment of abscesses involving the cricoid, especially when an osteomyelitis develops. This may also be necessary in extensive involvement of the thyroid. Mediastinitis and pneumonia may be sequelae and terminal events in extensive involvement, and it is to avoid these complications that radical procedures sometimes become necessary."

With these two opposing thoughts in mind, we must take our choice and institute the treatment most suited to the individual case.

CASE 1.—T. B., male, at the time of his illness was 72 years of age. During the Christmas holidays he had had the usual round of highballs, cocktails, etc., and on New Year's Day, 1935, had a little dizzy sensation and thought he might have an upset stomach. He intended taking some soda water, but noticed when he attempted to swallow that he strangled a little. The next morning, not feeling "up to snuff" he had breakfast in bed; but, when he attempted to swallow he noticed he strangled severely and coughed violently. He realized that his swallowing was so interfered with that he could not eat his breakfast. Every mouthful of food seemed to get into his trachea instead of going down his throat. I tried him with water which seemed to go down his larynx and which provoked a coughing spell in which he coughed up black pus. I found nothing wrong with his larynx except an edema of the arytenoids. Phonation was perfect, but there was some sluggishness of the epiglottis. The left side of the velum palati lagged a little. Paralysis was considered, but did not explain the tremendous amount of black foul-smelling pus which was present. I thought of a diverticulum of the esophagus, and since he had eaten considerable meat and fish during the holidays, I thought perhaps he had some foreign body lodged in the esophagus. We tried numerous positions to allow him to swallow, but a spillover into the trachea occurred with each attempt. We instituted rectal feeding and decided upon a waiting course. His swallowing did not improve, so the esophagus was investigated. An esophagoscope was passed on the ninth day, and no obstruction or diverticulum was found. We then knew we were dealing with a paralytic condition. A feeding tube was then inserted through the nose and left in about one week. It was removed occasionally to test his ability to swallow. A neurologist called in consultation located the central lesion as occlusion of the posterior inferior cerebellar artery, which he seemed to think was a spastic occlusion, and not a hemorrhage. He was encouraging about improved swallowing. Accordingly, the feeding tube was removed the twentieth day, after being in-dwelling eight days, and the patient was gradually taught to swallow again. Within two weeks he was able to swallow most soft foods except mashed potatoes. For some reason he was unable to swallow finely ground foods. His improvement was steady except that he continued to clear his throat constantly, and would occasionally spit up this foul black pus, the source of which we were never able to determine.

The vocal cords apparently were normal, but the arytenoids remained swollen all of this time. There was never any discomfort in the throat itself. On about

March 24 he developed a herpes zoster about the left face, neck and shoulder; and coincidental with this there was an increase in the clearing of his throat. During this time he suffered several dyspneic attacks, most of them at night. The swelling of the arytenoids increased and there was some edema of the cords and subglottic area. After one severe attack of dyspnea in the middle of the night, it was decided not to wait to do tracheotomy until it became a matter of emergency, to be done in the middle of the night without the aid of aseptic surroundings, etc. Therefore, on March 29 a tracheotomy was performed under local anesthesia, from which recovery was uneventful. This was during the time of the herpetic eruption and the swelling within the larynx almost completely obstructed the airway, so that the voice was hoarse at best. For a period of about eight months there was little or no improvement in the larynx. The patient continued to wear the cannula, and had a weak, though effectual voice, but no airway adequate for his needs. There was drainage below. The cords and subglottic space were intensely swollen. At this time, Nov. 5, 1935, a second operation was done through a high collar incision, with the hope of draining a perilaryngeal abscess. I dissected up behind the larynx in the common wall between the larynx and the esophagus, but never could locate any accumulation of pus in that region. However, there was a submucosal abscess above the tracheotomy cannula which was drained at this time. Considerable pus continued to drain from this locality for several weeks. A skin infection in the neck complicated the recovery; this, however, healed in due course of time, and since that time, there has been improvement in the larynx. The airway has become larger, the cords have become less edematous, the arytenoids have again become movable, but there is not yet sufficient airway to permit the removal of the tracheotomy cannula.

It has now been five years since the onset of this condition. It is my opinion, and that of consultants who have seen this case, that this was primarily a paralytic condition resulting from closure of the posterior inferior cerebellar artery, and that the osteochondritis of the larynx was the result of the herpetic lesion. We have never yet been able to explain the presence of the foul black pus that was present at the very onset. This man continues in excellent health and is able to carry on his business perfectly. He is now 77 years old, and goes everywhere, breathing through his tracheotomy cannula.

Case 2.—B. D., a young woman, aged 19 years, was in an automobile accident, Feb. 7, 1938. She was riding on the back seat of an overcrowded car when the wreck occurred and suffered lacerations to the skull and forehead. Her most serious injury, noted at the time, was a ruptured bladder. If the injury occurred to the larynx it went unnoticed in the presence of the severity of the symptoms from her ruptured bladder. She was admitted to the hospital and suprapubic drainage established. She had an extravasation of the urine into the pubic area, and a cellulitis of the thighs followed. She was in a most desperate condition for perhaps two weeks. During part of this time a Wangensteen continuous suction tube was used. The tube was in-dwelling from Feb. 9 to 18. A cough developed on Feb. 10, for which the patient was given terpin hydrate with codeine. She continued coughing throughout the recovery period and complained particularly of a severe sore throat for the first time on Feb. 17. The Wangensteen suction tube was removed on Feb. 18, after which she was able to eat soft diet. Recovery from her bladder condition was steady and continuous. On March 8, twenty-eight days after her injury, her sore throat became worse and she was hoarse and restless. During the nights following she was given luminal and codeine for the cough and

restlessness. On the nights of March 10 and 11 she had attacks of dyspnea which were mistaken for asthma by the interne, who administered repeated hypodermics of adrenalin, morphine and atrophine. This was the most dangerous, hazardous therapy which could have been thought of under the circumstances. During this time the dyspnea was growing steadily worse.

On the morning of March 12 at which time I first saw her she was in a desperate condition, cyanotic and struggling for air. I inserted a No. 5 bronchoscope. Her dyspnea was relieved immediately and her straining efforts for air were no longer necessary. I proceeded with an orderly tracheotomy at the level of the fourth ring, with the bronchoscope in place. The condition of the larynx was as follows: immense swelling of the entire laryngeal orifice and lumen; the structures could not easily be made out; the pyriform sinuses were obliterated and the arytenoids could hardly be seen; the bronchoscope had to be inserted forcibly into the larynx, whose lumen was entirely closed by swelling. Careful postoperative care was given because of the fact that she had no airway above the tracheotomy cannula. Healing took place around the cannula, and in due time the fistulous tract was complete. This young woman left for California soon after without my consent, and further attention could not be given to her abscessed larynx.

She has recently returned, and on Aug. 29, 1940, a direct laryngoscopy showed the larynx to be partially collapsed, with a lumen that would admit only a 3 mm. bougie. There still remains some edema, and it is possible that some infection of the cartilage is still active. By direct laryngoscopy and by palpation it is not possible to determine any indication for surgery on the larynx. It is my hope to follow this case through, and if possible, in time to evacuate any subperichondrial accumulation of pus. After healing is complete an attempt at dilatation or surgical treatment of the laryngeal stenosis will be undertaken.

CONCLUSIONS

These two cases are unique in their identical courses. It is possible that both resulted from the in-dwelling feeding tube, although we have ample evidence to doubt this. The patient in Case 1 was seen by several outstanding consultants, and it was the consensus of opinion that the herpes zoster produced the ulceration and subsequent necrosis of the laryngeal cartilages. Case 2 could easily have been a necrosis resulting from the Wangensteen tube. It is significant, however, that the cough and complaint of sore throat began almost too early to be the result of necrosis caused by the in-dwelling tube. It is very likely that the larynx in this case was injured at the time of the other injuries sustained in the automobile wreck.

SUMMARY

It is my opinion that laryngeal abscesses of this character come on very slowly following a trauma, and no doubt some cases have developed and run on to a fatal termination without being recognized, perhaps after patients have been dismissed and returned home

from the hospital. Certainly, tracheotomy is a life-saving procedure. The wearing of the tracheotomy cannula is apt to be prolonged; many months, even years, will elapse in some cases before normal laryngeal breathing can be re-established.

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XLVIII

TREATMENT OF BACTERIAL MENINGITIS OF RHINOGENIC ORIGIN*

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In 1936[1] we reported 19 cases of spheno-ethmoiditis with meningitic symptoms. Of this series, six patients who had a serous meningitis as a complication recovered completely, after a prompt intranasal spheno-ethmoidectomy. The remaining 13, including all cases of bacterial meningitis, succumbed to their infection. At that time we urged immediate surgical intervention as soon as symptoms of meningeal irritation appeared because this offered the only hope of recovery. When demonstrable bacterial invasion of the meninges complicated infection of the sinuses, surgery had proved futile. This failure was due to inability to cope with the meningeal infection, although the primary sinal disease was eradicated. As a result, we advocated prompt removal of the primary focus before fatal bacterial meningitis ensued.

With the advent of chemotherapy, it became possible to go a step further and sterilize the meninges after bacterial infection had occurred. However, experience has shown that recurrence or persistence of meningeal infection is frequent if the primary focus in the sinus is not adequately drained. Such reinfection may occur in spite of most energetic chemotherapy supplemented by transfusion and antibacterial serum. This failure to obtain a permanent cure without surgery is understandable if the histopathology of the sinus infection is studied. In a recent communication[2] we illustrated the various intracranial pathways of infection from the ethmoid and sphenoid sinuses. The frequent occurrences of primary submucosal abscess in the sinus with foci of osteomyelitis in the adjacent basisphenoidal or ethmoidal bones were demonstrated. By serial sections the pathways of spread from the primary focus in the mucosa of the sinuses could be traced through vascular and lymphatic channels

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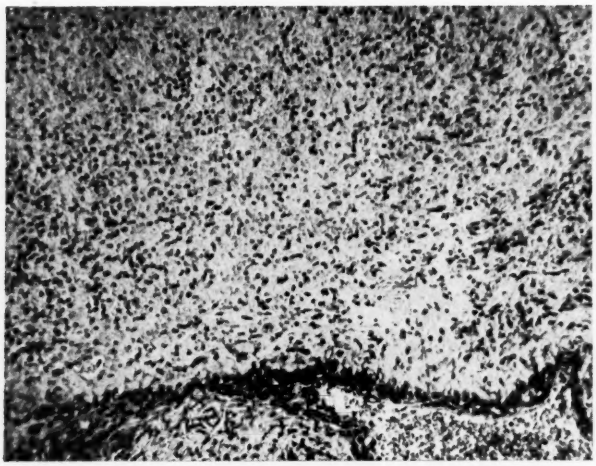

Fig. 1.—Mucosa of sphenoid sinus showing severe inflammation with areas of necrosis. Remnants of the epithelium with adherent pus are still attached to the mucosa.

to the meninges. We are of the opinion that in cases where healing of the sinus infection and bacterial meningitis takes place these infected lymphatic or vascular channels become sealed. We have microscopic evidence that when spontaneous healing occurs and the spinal fluid becomes sterile with or without chemotherapy, these portals of entry become sealed. This can be inferred by the replacement of the normal communicating vessels and channels between the dura and sinal mucosa with granulation tissue and fibrosis. Occasionally actual new bone proliferation can be seen as evidence of an attempt to wall off an avenue of invasion to the intracranial contents. These pathways of extension seem to be influenced by chemotherapy so that occlusion takes place. On the other hand, the original suppurative focus in the sinus mucosa as well as areas of bone suppuration are little affected by chemotherapy. Post-mortem study of paranasal sinuses and specimens of sphenoidal mucosa removed at operation have revealed persistent active suppuration with necrosis in spite of large doses of chemotherapy which had been sufficient to sterilize temporarily the spinal fluid (Fig. 1). If the chemotherapy be stopped or the dose diminished, there is the danger that the infection in the mucosa of the sinus or in the adjacent bone

may flare up and the meninges again become invaded. This actually happened on five different occasions (Lloyd, Seidlin, Trauhauft, Cuilla and Sacks). In these instances the initial bacterial invasion of the meninges was controlled by adequate chemotherapy after the offending organism had been cultured from the spinal fluid. On withdrawal of the drug, subsequent and more violent bacterial meningitis occurred which was finally controlled only after surgical drainage of the primary focus in the sinus was added to chemotherapy.

We are now reporting 19 additional cases of bacterial meningitis of rhinogenic origin. These have been observed at the Mount Sinai Hospital since the advent of chemotherapy. All have the following three features in common: (1) The primary focus was in one of the paranasal sinuses. (2) Each patient had bacteria in the spinal fluid on admission. (3) Each patient was treated with large doses of an appropriate sulfonamide. In addition 13 had the focus in the sinuses surgically drained and six were treated conservatively without surgery.

An analysis of our results in these 19 cases follows: (1) Six made complete recoveries after intranasal spheno-ethmoidectomy and chemotherapy. (2) Six were treated conservatively with chemotherapy, antisera and transfusions, and all died. (3) The remaining seven received both chemotherapy and surgery but nevertheless succumbed to their infection.

In all instances, the bacterial meningitis was present when the patient came under observation. In 17 cases a positive culture was obtained from the cerebrospinal fluid and the bacteria identified as pneumococcus in 11, as Streptococcus hemolyticus in four, as Staphylococcus aureus in one and as Staphylococcus albus in one. In two other instances the patients had received large doses of chemotherapy before hospitalization. Direct smear of the spinal fluid repeatedly showed gram-positive cocci which failed to grow on culture.

The rhinogenic origin of the bacterial meningitis and the suppuration in one of the paranasal sinuses as the primary focus of infection was proved by operation in 13 cases. Necropsy revealed gross and microscopic evidences of the pathway of spread from the sphenoid sinuses in two instances. In another, orbital cellulitis and periostitis substantiated the clinical impression of an underlying ethmoiditis. The diagnosis of purulent sinusitis as the source of infection was made in the remaining three instances of pneumococcal

meningitis on the basis of the clinical history and the intranasal examination. The chest was clear in each one and evidences of sinal suppuration were present. The frequently fulminating clinical course of rhinogenic bacterial meningitis may be profoundly modified and often masked by chemotherapy. Early indications of meningeal invasion are apt to be overlooked because of the masking and the antipyretic action of the drug. The prognostic significance of the spinal fluid findings had to be re-evaluated.

A résumé of a few illustrative cases is presented so that certain features in management and therapy can be stressed.

CASE REPORTS

CASE 1.—(No. 438282.) A. L., an adult male, had an acute upper respiratory infection two weeks before admission. For forty-eight hours before admission he suffered from severe headaches, vomiting and a temperature of 104° F. Examination on admission revealed clinical features of bacterial meningitis with disorientation and photophobia. Intranasal evidence and roentgen examinations showed suppurative right ethmoiditis to be present. Lumbar puncture yielded purulent fluid under increased tension, a pleocytosis of 5,600 cells, 95 per cent polymorphonuclear neutrophiles and gram-positive cocci on smear, which proved to be pneumococcus type XIX when cultured. The culture of the pus from the right ethmoid region was also pneumococcus type XIX. An intranasal ethmoid operation was advised, but the patient was treated with sulfapyridine which attained a level of 7.1 mg. per 100 c.c. in the blood and 4.6 mg. per 100 c.c. in the spinal fluid. After one week of therapy a spinal block developed and cisternal punctures were performed daily, 8 c.c. of air being injected into the spinal system and free fluid containing 500 cells with 40 per cent lymphocytes being obtained. On the sixth day after chemotherapy was begun the spinal fluid became sterile and 85 mg. of sugar per 100 c.c. was present. The patient was rational, afebrile and had but few residual signs of meningeal irritation. Cisternal punctures were continued daily and on the fifteenth day the spinal fluid was clear, containing only 41 cells. The temperature now rose to 102° F. Two days later he suddenly developed severe occipital pain and marked rigidity of his neck, and became totally irrational. His temperature rose to 105° F. At this stage his spinal fluid contained 50 cells. The following day a bilateral intranasal spheno-ethmoidectomy was performed and a large amount of mucopus was encountered in both spheno-ethmoid sinuses, more on the right. Both sphenoid sinuses were extensively pneumatized. The mucosa was stripped away from the roughened bone on the posterior wall. The patient became afebrile three days later, but he developed some hematuria from precipitation of the sulfapyridine. This subsided after the drug was discontinued, and the patient made an uneventful recovery.

This case illustrates several interesting features. Although the initial bacterial invasion of the meninges was readily controlled by chemotherapy, the primary focus in the sinuses was not checked. Sinus suppuration continued and reinfection of the meninges occurred until the sinuses were adequately drained surgically. We have

had four similar experiences in our present series. The efficacy of the drug is undoubtedly enhanced by removal of the suppurative primary focus.

The occurrence of spinal block is not of such grave significance today as before chemotherapy. Furthermore, improvement in spinal fluid findings is of less prognostic import than formerly, in that it does not necessarily indicate control of the meningeal infection. A clear spinal fluid with a minimal pleocytosis does not exclude the possibility of a latent bacterial infection. The bacteria may be inhibited by the sulfonamides and growth in the culture may not occur for three days. Some cultures which were sterile for forty-eight hours yielded bacterial growth in seventy-two hours.

CASE 2.—A. S., a 31-year-old male, with the history of profuse nasal discharge, two weeks after swimming was admitted to the medical service because of excruciating parietal headache and fever of 102° F. On examination he appeared apathetic, drowsy and in extreme pain from severe headache. A nose and throat examination revealed the presence of a bilateral suppurative spheno-ethmoiditis. A lumbar puncture showed clear fluid containing only one lymphocyte, but the culture yielded a pneumococcus type XII in one flask. The following day the spinal fluid was still clear, but the Pandy test was four plus. The patient was placed on sulfapyridine therapy, both intravenously and orally. The fever subsided but the intense unyielding headache persisted. After several days the temperature rose sharply, and signs of meningeal irritation appeared. Eight days after admission, because of lack of response to chemotherapy, a bilateral spheno-ethmoidectomy was performed. An empyema of both sphenoid sinuses was found. The mucosa appeared gangrenous and necrotic. This was stripped from its bony attachment. The pathological report showed acute purulent inflammation with areas of necrosis and focal periostitis. Postoperatively there was immediate relief of the headache. Temperature became normal, and the patient generally improved. A right lateral rectus developed on the fifth postoperative day. Chemotherapy was stopped and the patient made a prompt uneventful recovery. The sixth nerve palsy cleared up completely.

This case again illustrates the persistence of active suppuration in the sphenoid sinus in spite of large doses of chemotherapy and good nursing care with an attempt to promote drainage by conservative treatment. The areas of necrosis in the sinus mucosa testify to the lack of response to chemotherapy. The occurrence of the sixth nerve paralysis five days after operation was not necessarily indicative of an intracranial complication, but more likely represented a localized dural irritation in an extensively pneumatized, markedly inflamed sphenoid sinus. Reliance placed on the efficacy of surgical drainage rather than on further chemotherapy prompted us to stop the sulfapyridine and observe the patient. The sixth nerve paralysis subsided and the patient made an uneventful recovery.

CASE 3.—A. S., a 17-year-old male, was admitted to the laryngological service with a history of an acute upper respiratory infection two weeks previously. Five days after the onset, he developed severe pain in the right eye and had frontal headache with a temperature of 102°. A diagnosis of acute sinusitis had been made and he had been given 90 gr. of sulfanilamide daily. The fever persisted for five days and then the patient had a chill and rise of temperature to 106°. A consulting physician advised quinine and increased doses of sulfanilamide. The fever subsided and remained normal for five days, during which he received chemotherapy. The sulfanilamide was discontinued for twenty-four hours. He became delirious and had a shaking chill. The temperature was 105.6° on admission. He appeared critically ill, toxic, lethargic and disoriented. The sclera were subicteric and the lips were cyanotic. He had a rigid neck, bilateral Kernig and photophobia. He had ankle clonus and absent cremasteric reflex on the left. The right ocular fundus was blurred. The nasal septum was markedly deviated to the right, occluding the nasal chamber; thick pus was present in the anterior ethmoid region and nasopharynx. There was questionable tenderness over the floor of the right frontal sinus. X-ray showed clouding of all sinuses on the right. The spinal fluid was clear, yellow and contained 300 cells, 60 per cent polymorphonuclears, 40 per cent lymphocytes, and the Pandy was four plus. An external frontal operation was performed promptly while a continuous intravenous administration of saline solution and sulfanilamide was given. Foul-smelling pus was found in the right frontal sinus. The inner table of the sinus appeared normal but at its junction with the anterior wall, osteomyelitis was found. Thick pus was found in the diploe of the frontal bone as far back as the parietal region. A large epidural abscess was uncovered. There was an area of necrosis over the frontal lobe, overlying an abscess. The patient died within twenty-four hours in spite of large doses of chemotherapy and transfusion.

This case is cited especially to stress the sudden violent meningeal flare-up after withdrawal of the chemotherapy. There is little doubt that the osteomyelitis and epidural abscess had existed for some time before the fulminating meningitis became apparent. In spite of the extensive disease found at operation, no clinical indication of its existence was manifest until the drug was stopped. The classical signs of osteomyelitis such as edema, tenderness, chills and fever were absent during chemotherapy. It must be assumed that the drug altered the clinical course of the illness and masked such symptoms and signs as might have made the need for surgery very obvious. The drug failed both in arresting the infection in the frontal sinuses and in preventing the ensuing complications of osteomyelitis, meningitis and brain abscess.

There seems to be a distinct hazard in the indiscriminate use of chemotherapy in the treatment of acute suppurative sinusitis. This is particularly true when sole reliance is placed on chemotherapy and the other established methods of promoting drainage are omitted. The drug not only may prove ineffective in curing the

sinus infection but may so mask the symptoms that complications will be unrecognized until well advanced.

The antipyretic action of the drug may lull one into a sense of security which is often not justified. The principles of adequate surgical drainage when indicated should not be abandoned in the hope of cure by chemotherapy. Once adequate drainage has been established, chemotherapy has proved effective in preventing complications and hastening recovery.

CASE 4.—R. S., a 45-year-old diabetic female, was admitted to the laryngological service with the history of an acute upper respiratory infection two weeks before admission. For the next ten days she had had severe frontal headache and nasal obstruction. Twelve hours before admission the nasal discharge stopped and the headache became worse. Upon examination the patient was drowsy, had a stiff neck and a temperature of 104°. The septum was markedly deviated to the left; there was a large amount of pus in the right middle meatus, a small amount on the left side and thick pus in the posterior choana. The culture showed pneumococcus type III. The urine examination showed sugar four plus, acetone three plus, albumin three plus; blood sugar, 235 mg. Spinal fluid showed 5,000 cells, 76 polymorphonuclears, Pandy four plus. Gram-positive cocci were found on smear. Culture then of the spinal fluid showed pneumococcus type III. Blood culture on admission was pneumococcus type III. The patient was started on chemotherapy and operated upon on the day of admission. A bilateral spheno-ethmoidectomy was performed. An empyema with thick pus under pressure was encountered in the left sphenoid. There was edema of the mucosa in both sphenoids. An intravenous infusion of sulfapyridine, 0.6 per cent, with 10 per cent glucosa in saline solution was given. The first day, 16 gm. was given by this route; 33.5 gm. was given the first eighteen hours; 140,000 units of type III pneumococcus antiserum were given intravenously and 20,000 units intraspinally. After eighteen hours of therapy, the blood level of sulfapyridine was reported to be 15.7 mg. per 100 c.c. and the spinal fluid level 11.9 mg. per 100 c.c. After the first day, no growth was reported in repeated blood and spinal fluid cultures. The patient now developed anuria and the drug was stopped for twenty-four hours but fluid intravenously was continued. Rapid resumption of kidney function followed and the drug was administered again. After one week of treatment, the temperature was 101°, the hemoglobin fell to 55 per cent with erythropoiesis and the drug was again stopped. She was given a blood transfusion and recovered from the hemolytic episode. From this point on she made an uneventful recovery.

Both the bacteremia and the meningitis were cured by a combination of surgical drainage, chemotherapy and anti-pneumococcus serum. Whatever the portal of entry may have been, it became promptly sealed and further invasion of the meninges did not occur. The patient was given several doses of sulfapyridine before the operation was started and she showed no adverse effects from the operation.

We have been less fortunate in several other instances where intranasal operation was promptly performed in the presence of a fulminating bacterial meningitis. Some of these patients were ex-

tremely toxic, dehydrated and had a very rapid pulse with a high fever and in some instances were stuporous. Patients in this condition do not tolerate surgery well even if it is performed rapidly and under local anesthesia. Their condition resembles surgical shock and an attempt should be made to overcome this before operating. Supportive therapy with transfusion and antisera should be given as indicated. We feel that large doses of sulfonamides should be administered promptly by the intravenous and oral routes if possible. The aim should be to combat the meningeal infection first and then attack the primary focus within twenty-four to forty-eight hours, or as soon as the general condition permits. If the meningeal invasion is so overwhelming that chemotherapy is ineffective, there is little likelihood that drainage of the primary focus will enhance the chances of recovery. This period of waiting is advisable only in those instances in which the patient's condition is so toxic and shock-like that operation will not be tolerated. However, general improvement in the patient's condition should not be used as an argument for delay of operative therapy.

SUMMARY

Nineteen cases of bacterial meningitis of rhinogenic origin were all treated with large doses of sulfonamides plus antisera and transfusions as indicated. Thirteen of these were treated surgically in addition to chemotherapy. Six of this group made complete recoveries by this combination of surgery and chemotherapy and seven died. All of the six patients who were treated by chemotherapy alone died.

Chemotherapy has proved invaluable in sterilizing the meninges after the initial invasion from a focus in a neighboring nasal sinus. Reinfection of the spinal fluid is to be expected unless the primary focus is adequately drained surgically. The sulfonamides alone have been found ineffective both in controlling the suppuration in the sinuses and in preventing the complications of osteomyelitis and brain abscess. The routine use of these drugs in the treatment of acute sinusitis seems to be inadvisable. The sulfonamides may mask the early clinical symptoms and signs of a serious complication which are apt to become manifest on withdrawal of the drug.

The use of chemotherapy in combination with surgery has yielded cures in cases of bacterial meningitis of rhinogenic origin. The patient in this type of case almost invariably succumbed before the advent of the sulfonamides. Patients who are desperately sick and toxic from an overwhelming infection of the meninges should

first be treated with sulfonamides and supportive treatment before operation on the sinuses.

In 13 of these surgical drainage of the suppurative foci in the sinuses was instituted in addition to chemotherapy.

121 E. 60TH ST.

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XLIX

OSSIFYING FIBROMA OF THE UPPER JAW*

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AND

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Ossifying fibroma is a form of benign fibro-osseous tumor, most frequently found in the upper and lower jaws. Phemister and Grimson[1] reported thirteen cases and detailed two of Montgomery's cases, in which the age ranged from 8 to 69 years. Of these fifteen patients, four were above 40 years of age. All of our patients were negroes, but in view of the occurrence of this tumor in white patients reported in the literature, we are not prepared to state that the disease is more common in negroes.

The genesis of ossifying fibroma is not well understood in spite of the fact that the lesion is probably less rare than the sparsity of the case reports would indicate. There are certain leads to the nature of the tumor given by its histologic appearance, but it is impossible to state with certainty whether the lesion is truly a spontaneous benign neoplasm, a reaction peculiar to membranous bone to trauma and infection as suggested by several authors, or a "growth disturbance" as suggested by Eden.[2] It is also possible that the ossifying fibroma, like the giant-cell epulis, represents a developmental cell derangement of the dental periosteum incident to the completion of the permanent tooth cycle in the deciduous teeth.

These tumors in the past have been erroneously classed as central fibrospindle-cell sarcomas, but the fact that the ossifying fibroma tends to differentiate into well-formed bone, and to reach a static phase of development is itself an indication that the tumor is benign. Clinical experience has proved that metastasis does not occur, but the tumor may recur if not completely excised. More recently this tumor has been named central fibroma, sclerosing fibroma, and osteoid osteoma. Some authors prefer to call younger, more fibrous

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*Read before the Southern Section of the American Laryngological, Rhinological, and Otological Society, at Atlanta, Ga., Jan. 23, 1942.

tumors ossifying fibromas, and to call the older more osseous ones osteomas.

The symptoms of ossifying fibroma are chiefly related to the growth of the tumor. Pain is not a characteristic symptom, and for this reason the tumor may and probably does grow for years, especially in the antrum, before it is noticed.

Roentgenographically, malignancy may be ruled out by the smooth contour of the ossifying fibroma. The absence of multiocular rarefied areas distinguishes this tumor from giant-cell tumors, cysts, and adamantinomas.

The treatment of choice is surgical excision. According to Geschickter and Copeland:[3] "In adults with slowly growing tumors of this type the lesion should be watched rather than operated upon during the period of growth. The preferable treatment is careful excision, with cauterization of the surrounding region, during a period of quiescence. Even with recurrences such treatment should be tried when more radical removal means mutilation. Up to the present time, the cases in this series treated by irradiation have not responded favorably."

On the other hand, Arons[4] successfully treated one case of ossifying fibroma of the superior maxilla by X-ray. The first diagnosis in this case was osteogenic sarcoma. We do not believe that any of the three cases treated by surgical excision reported here had more tissue removed than was actually necessary to get rid of the tumor.

The excised tumor is coarsely nodular, circumscribed, and either fibrous and gritty to the cutting knife or bony hard, depending upon the age of the lesion. Microscopically, the younger ossifying fibromas are composed of loose proliferating fibrous tissue containing spicules of poorly calsified bone or osteoid tissue. These spicules are not often club shaped like regenerative new bone as it occurs in bone derived from cartilage, but it is evident that they represent new bone formation because older lesions have calcium laid down in spheroids or in homogeneous diffusion throughout the spicules. In the younger lesions there are active osteoblasts along the edges of the osteoid spicules, and there are frequent foci of giant cells of the foreign body type which are engaged in osteoclastic activity.

The histologic appearance of these younger lesions suggests a relationship to osteitis fibrosa of long bones, but since this particular microscopic picture is not specific, it must be admitted that the

Fig. 1.—Case 1. Postoperative scar of incision, from corner of mouth to top of malar bone.

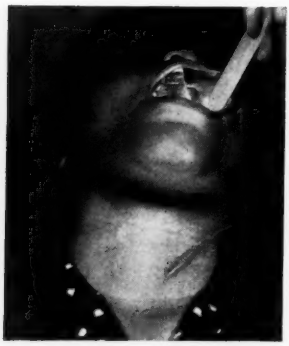

Fig 2.—Case 1. The healed wound. The palate was split in the midline, although the angle at which this photograph was taken makes it appear to be off the midline.

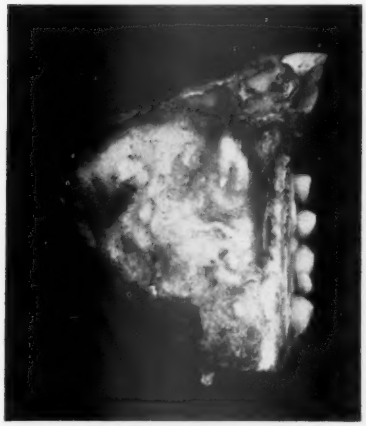

Fig. 3.—Case 1. Gross specimen.

only likely relationship is that both lesions represent an active attempt on the part of bone to defend itself from some injurious agent or metabolic defect. The presence of giant cells is another indication of reaction of bone to unknown stimulus. Here the relationship to osteitis fibrosa ends, because in none of the reported cases of ossifying fibroma is there any indication of generalized osseous fibrosis. Furthermore, Klassen and Curtis[5] after running phosphorous and calcium metabolism tests in three cases of ossifying fibroma found both normal. Phemister and Grimson[1] found serum calcium and phosphorous normal in four cases. This would indicate that there is no imbalance of the general skeletal mechanism.

In our opinion the analogue by some authors of ossifying fibroma of the jaw with benign osteomas of other flat bones of the skull is not well taken, because we do not believe that the lesions are histologically comparable. The osteoma of the cranial vault, for instance, seems even in soft formative stages entirely destined to form new bone and has none of the sclerosing fibrous reaction or giant cells seen in softer ossifying fibromas of the jaws.

The relationship of ossifying fibroma to epulis of the giant-cell type is also suggested. The usual location of occurrence of the two tumors is similar in that they both occur in the region of deciduous teeth and both tend to appear early in life. A dissimilar feature of these two tumors is that the giant-cell epulis grows peripherally,

Fig. 4.—Case 2. Patient before operation.

but the ossifying fibroma is a central tumor and does not grow peripherally. The analogue between these two tumors is not definitely broken by this supposed difference, because the very difference in location of the two tumors may account for differences that exist in the histologic picture, and it is entirely possible that since the epulis is supposed to arise from dental periosteum, the ossifying fibroma may also arise from dental periosteum that lies deep in the dental ridge, and therefore gives rise to a central tumor. This point is made clearer by the demonstration, in Case 1 of this series, of a canine tooth embedded in the substance of the tumor. Other authors have noted the absence of certain teeth over an ossifying fibroma, but as far as we are aware, they have not demonstrated a tooth in the substance of the tumor. We have done so in only one of four cases, but it is not essential to the theory that these tumors like the giant-cell epulis may arise from dental periosteum, that such an embedded tooth must be found.

In older lesions the histologic picture becomes less exciting to the imagination. Development of dense bone simply proceeds in an orderly fashion with the formation of hematopoietic and fatty marrow in some cases.

In our series, the patient in Case 2, a child, had a soft lesion that presented the early fibrotic histologic picture. The patient in Case 1, a young adult female, had a harder lesion with better calification and more bone formation. In Case 3, the bone formation was complete and dense with development of hematopoietic marrow.

A fourth case, observed on the service of our colleague, Dr. R. Lee Clark, did not fit the usual clinical or histologic picture of ossifying fibroma.

Dr. John C. Herthorne, Pathologist, Mississippi Baptist Hospital, Jackson, Miss., reported the following: "In this particular case, the patient was 52 years old and the tumor had been removed twice elsewhere during the previous six years. Evidently the tumor had been present for some years before the first operation. The microscopic picture was that of an ossifying fibroma in the fibrous stage with very little bone formation, the sort of microscopic picture one would expect in the tumor as it occurs in children. In addition, the large number of osteoclastic giant cells led us to consider the possibility that this tumor was a true giant-cell tumor of the jaw, derived from the region of Meckel's cartilage, but this possibility was discarded in view of the absence of any cystic degeneration, and the unmistakable fibrous and osteoid structure in many areas. The tumor in this case was enormous, and had evidently been influenced in part by the two incomplete removals."

CASE 1.—The patient was a negro woman, 33 years of age and of unusual intelligence. The history was of a swelling externally, below the left eye and in the hard palate, which had begun about ten years before and had grown gradually. The patient thought it had grown rapidly during the last year; perhaps, perhaps not. There had never been any pain or soreness. There was a marked bulging below the eye externally, above the teeth inside the lips, and of the entire hard palate on the left side. The swelling did not extend beyond the midline of the hard palate. This patient was referred to me by Dr. Brister Ware, who ligated the external carotid artery a few days before the tumor was surgically attacked. The technic of the operation was that of resection of the upper jaw. A part of the floor of the orbit was left, however, and the pterygoid processes were cut through at the base of the skull instead of being separated from the superior maxilla. The entire operation was done under local anesthesia without pain or bleeding. One feature about this case that interested me was that before the operation on the jaw, but after ligation of the external carotid, the patient noticed that the vision was not clear in the left eye. On July 9, twelve days after ligation, six days after the jaw operation, the patient noticed a queer feeling in the left eye and then found that the vision was gone. There was at this time an acute diffuse retinitis in this eye over the entire fundus, but especially marked in and about the macula region. The arteries were not completely filled with blood and the nerve head was pale. The vision on July 22nd was moving objects. On Aug. 4, there was a white exudate accompanying the retinal vessels in all directions,

Fig. 5.—Case 4. Preoperative view of patient described by Dr. John C. Henthorne.

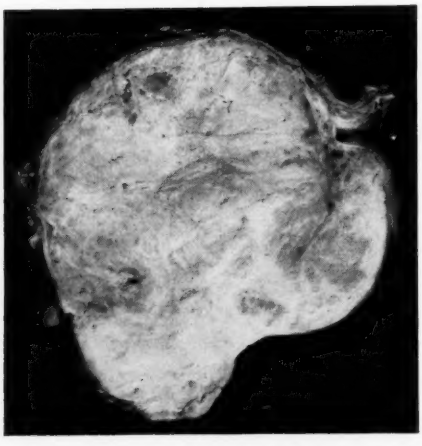

Fig. 6.—Case 4. Gross pathologic specimen.

greater at the disc and diminishing toward the periphery and there was a star at the macula. On Aug. 11, the vision in the left eye was 20/100. The retinal lesions have always resembled those accompanying a malignant hypertension, but there were no lesions in the right eye and a medical consultant found no blood vessel pathology and no hypertension. At the present time the disc is quite pale and the vision is still 20/100.

CASE 2.—A male child, age 12 years, of Sinegambian lineage was admitted to the Baptist Hospital in July, 1941. His complaint was that of a mass in the upper right jaw (Fig. 4). This swelling had begun about six months before and the growth had been rather rapid. Skin was movable over the mass, and the nose and orbit were not involved. The right external carotid was ligated on July 9, 1941, and two days later the tumor operation was done under ether anesthesia. The incision was from the right cheek to the corner of the mouth and along the alveolar process above the teeth. The skin flap was turned back and the periosteum elevated. The tumor mass was found to fill the antrum completely. The tumor was removed and the tissue curetted down to healthy bone. Iodoform packing was allowed to remain for ten days. The patient was discharged in good condition. Neither X-ray nor radium was used.

CASE 3.—A colored female, 41 years of age, entered the Hospital on Sept. 9, 1941, complaining of a swelling in the right upper jaw which had only been present about four months. Examination revealed a mass involving the right posterior alveolar ridge of the maxilla. The mass measured about 2½ inches in length and 1½ inches in diameter. After ligation of the external carotid artery on Sept. 9, the bony tumor was removed under sodium pentothal intravenous anesthesia. The bone and tumor were chiselled away including the molar teeth, leaving no floor to the maxillary sinus. The sinus mucosa, however, was left intact and it was found to be normal. The area was packed with iodoform gauze which was removed in two days. On Sept. 14, the patient was discharged in excellent condition.

EYE, EAR, NOSE AND THROAT CLINIC.

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ENCEPHALOMENINGOCELES IN THE NASAL CAVITIES

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Congenital herniations of the brain are relatively uncommon. These herniations produce tumors which are usually located in or near the midline from the nasofrontal region anteriorly to the occiput and foramen magnum posteriorly. They have been reported by a great variety of titles but the most descriptive and accurate is encephalomeningocele, as both neuroglia tissue and meninges are present. They are often found at the sites of the fontanelles according to Mood.[1] He lists the occipital, frontal, sphenoidal and mastoid as fairly common sites. Less frequently, they penetrate the cribriform plate of the ethmoid, the foramen cecum, an enlarged foramen magnum, or one of the various suture lines.

The usual classifications given by authors reporting these cases have been those of von Meyer[2] and Heineke.[3] Von Meyer's[2] first sincipital type is the nasofrontalis which leaves the cranium between the frontal and nasal bones producing a tumor in the median line in the region of the glabella. His second type is the nasoethmoidalis which emerges between the frontal, nasal and ethmoid bones and appears at the junction of the cartilaginous and bony parts of the nose. The third type described by von Meyer[2] is the naso-orbitalis which has its pedicle passing through one of the orbital fissures into the orbit or into the sphenomaxillary fossa becoming visible below the zygoma.

Heineke's first type of basal hernia may emerge through any part of the cribriform plate into the nasal cavity. The second is the sphenopharyngeal which leaves the cranium through an opening between the sphenoid and ethmoid bones and hangs in the choanal region of the nasal cavity. The last type may emerge into the nasopharynx through defects between the sphenoidal and occipital bones.

Most writers have described these encephalomeningoceles as cysts containing cerebral tissue and cerebrospinal fluid connected, in many cases, with the ventricular system of the brain by pedicles

that may be large or small. The brain tissue of the cyst is usually covered with meninges and the walls are fibrous tissue enclosed by skin or mucosa.

The embryological origin has been the subject of much dispute as pointed out by Mood.[1] Guthrie and Dott[5] felt that they were formed during very early embryonic life before primitive elements of the skull or cribriform plate appear, by the outward growth of a small bud of tissue from the anterior cerebral vesicle. They theorized that at a later stage when the membranous skull is laid down, this bud of tissue may prevent the complete separation of the ectoderm from the neuroepithelium. A foramen may remain through which the encephalocele retains its connection with the brain. The small ones are frequently not connected with the brain at all but are found by accident in microscopic examination of turbinate tissues.

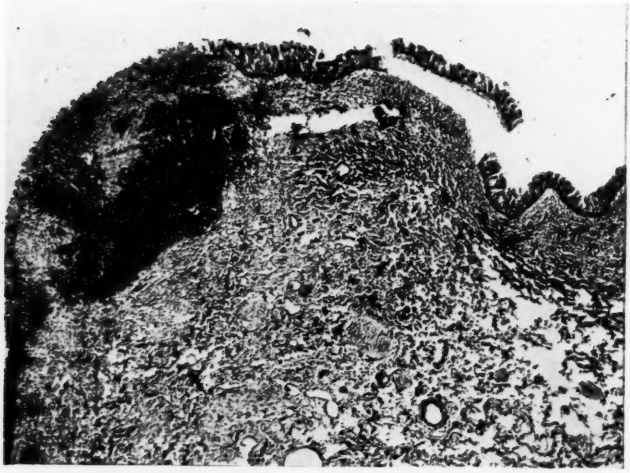
As our subject concerns only those encephalomeningoceles found in the nasal cavities, Rawling's[8] warning is not entirely pertinent. However, it is a valuable admonition in dealing with these interesting tumors. He warned that if the tumor is large with adherent skin but very little pedicle, and if it is firm with little fluctuation and no pulsation, and if there are symptoms of brain irritation, then brain matter is almost certain to take a large share in the formation of the tumor.

Lampert,[7] of Moscow, reviewing the literature of extra cranial tumors containing brain tissue in 1924, found 244 cases. Only 9 per cent of the patients lived to a mature age and there was a mortality of 50 per cent with surgery. The vast majority of these cases, however, were not intranasal encephalomeningoceles. The latter are possibly quite as common but are rarely reported, and are much more apt to be fatal in early life. He included gliomas in his general group and they should not be confused with encephalomeningoceles as pointed out by C. V. Weller in a personal communication: "The glioma is a neoplasm without developmental error while the encephalocele is a developmental error without neoplasia."

Lampert made the interesting observation that the older the tumor the more connective and less brain tissue there is to be found. In the fetus, the tumor is aptly to be mostly brain tissue with meninges and a small amount of connective tissue separating the brain from the skin or mucosa.

In my search of the literature, I found a paucity of case reports dealing with the intranasal variety. As late as 1932, Hallerman[8]

Fig. 1.



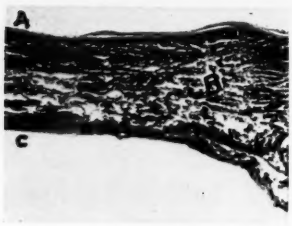


Fig. 2.

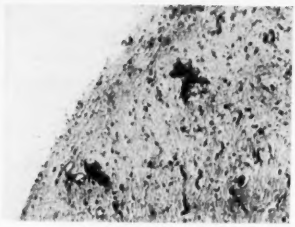


Fig. 3.

Fig. 1.—Section shows the wall of the encephalomeningocele covered externally by nasal mucosa. An intermingling of brain tissue is found in the deeper structures. There are chronic inflammatory infiltrations of the submucosa. The dark areas are hemorrhages in the submucosa following surgical manipulation.

Fig. 2.—Thinner section of the wall of the encephalocele covered externally by: *A.* Denuded nasal mucosa. *B.* Dura. *C.* Endothelium of the dura.

Fig. 3.—Portion of the gyrus of the cerebral hemisphere contained in the encephalomeningocele. Marked gliosis and atrophy is found. There is marked passive congestion and hemorrhage.

could find only eight cases in the literature. All had been reported prior to the advent of chemotherapy and all resulted fatally with the exception of one case. This had been reported by Fenger[4] and was a large sphenomaxillary encephalocele in an adult. It was operated upon successfully in 1895, by resecting the superior maxilla and ligating the pedicle.

I found one other authentic case, an intranasal tumor in a 3-day-old infant successfully removed by snare and reported by Rocher and Anglade[9] in 1924.

Most of the other patients whose cases were reviewed died of meningitis at an age varying from 3 days to 3 years. There were two exceptions. One reported by Nager[10] had a trans-sphenoidal tumor, lived several months after operation, and died at the age of 13 years. Another seen by Wiedel died at 18 years of age after a frontal sinus surgical approach. Doubtless, there have been many not recognized or reported. Possibly some of these have recovered following removal of the tumor.

There can be no doubt that there have been surgeons who have removed and discarded innocent appearing intranasal polyps and tumors only to have the patient promptly die of meningitis. Some of these tumors probably have been small encephalomeningoceles with pedicles leading through cribriform dehiscences directly to cerebral ventricles.

The prognosis has been so hopeless that most writers have advised against any interference with the intranasal type.

The differential diagnosis of this tumor is often extremely difficult as it may closely resemble a polyp or fibroma.

They may pulsate synchronously with the respiratory and circulatory oscillations of the brain, as pointed out by Furstenberg.[11] Compression of the internal jugular veins may cause the tumor to become firm and tense. But if it is old and connective tissue predominates, none of these phenomena will occur. Instead, the thick walls will even obscure the fact that it is cystic. They have been described as pale or dull red, smooth and glistening or, in some cases, eroded.

Diagnostic puncture or biopsy is, of course, just as dangerous as removal, as the contents of the cyst may communicate with the ventricles.

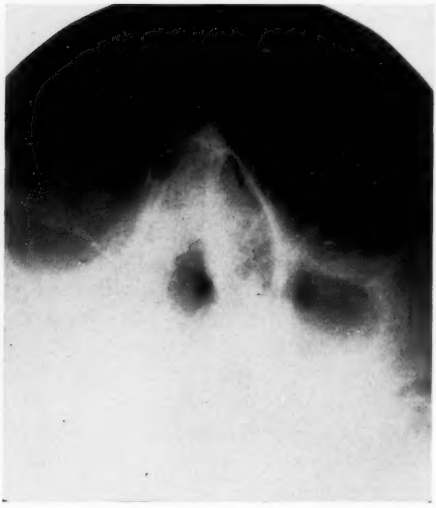


Fig. 4.—Roentgenogram illustrating cloudiness of left antrum into which the tumor had grown. Left nasal bones show an apparent defect.

If the correct diagnosis is to be made, the surgeon must be wary of the unusual history of cerebrospinal rhinorrhea. This rhinorrhea is a fairly constant finding. The fluid probably escapes around the edges of the pedicle at the cribriform dehiscence or, in case of erosion, through the dependent portion of the tumor.

The surgeon should trace the pedicle of any suspicious tumor before removal, and all tissue that is removed should be examined by the pathologist. If brain tissue is found in the sections, prompt chemotherapy may prevent a fatality. Because of the difficulty in diagnosing these tumors, no traction should be used in removing any nasal tumor or polyp that leads into the superior meatus.

If surgery seems absolutely necessary, the operator must make a difficult decision as to the best method of removing an intranasal encephalomeningocele.

The Naffziger[12] type of approach may be made by rhinologist and neurosurgeon through the frontal bones and above the sinuses. The dura and brain could be lifted from the cribriform plate and the pedicle ligated and cut. This approach would seem to offer the best chances of a cure and is successfully used by neurosurgeons to control cerebrospinal rhinorrhea due to cribriform plate fracture.

If the pedicle is a very large one, however, there results a large cribriform dehiscence with brain tissue directly exposed to the nasal cavity. In most of the autopsies on these cases, the dura has been firmly attached to at least part of the rim of the cribriform dehiscence and this would add to the difficulty of ligation and removal. Sulfanilamide packing might be placed over the cribriform plate and left until thick resistant dura covered the dehiscence, or a skin or fascia graft might be used to close the opening. Lampert[7] suggested a bone graft with periosteum attached and urged frequent lumbar punctures to control the excess cerebrospinal fluid found in many of these cases.

An external ethmoid approach offers excellent visualization of the nasal side of the cribriform plate. If the pedicle is small, it might be ligated and cut without too much difficulty. However, closing the dehiscence would offer an exceedingly difficult problem.

The removal by snare wire, without traction, through the nasal cavity has one advantage. It leaves the cribriform opening blocked with fibrous wall which has been exposed to nasal secretions and which may have acquired some resistance to nasal infections. Its disadvantages include opening the pedicle to ascending infection and the tendency for recurrence.

CASE REPORT

Donald C., aged 13 years, was first seen on Nov. 1, 1940. His parents stated there had been difficulty in breathing through the left nostril since birth. For two years both nostrils had been completely blocked and distortion of his nose was developing to an alarming degree. For the past year, a tumor mass had been protruding from the left nostril and he refused to attend school and submit to the ridicule of other children.

At the age of 3 months, swelling of the left cheek appeared and the tumor began to protrude from his nostril in a similar manner. The protruding portion was removed without incident. The tissue was sent away for examination but the doctor who removed the tumor could not be contacted and the report was not available. On several occasions, during his life, he had lost consciousness for a day or two, ran a fever and seemed delirious, but these symptoms disappeared as quickly as they came.

He had a continuous drainage from the left nostril sometimes purulent, again clear, and described by the mother as mucus in character most of the time. He often used fifteen handkerchiefs a day. There were no other headaches or symptoms. His parents stated that in every other respect he was a normal bright child. The youngest in a large family of children living in extreme poverty and filth, he had developed a marked inferiority complex because of the appearance of his nose.

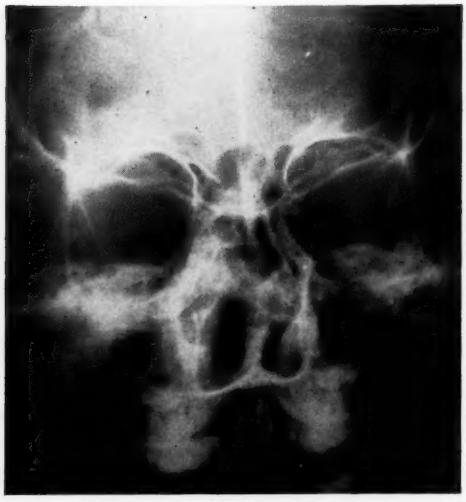


Fig. 5.—Film taken after removal of the encephalomeningocele showing the left antrum clear and revealing the complete displacement of the septum to the right. Neither middle turbinate bone has developed.

Examination revealed an alert, cooperative boy whose features were thick and coarse. There was marked widening of the nasal bridge and distortion of the left side of the nose. The left nostril was completely filled by a fibrous, questionably cystic tumor which was protruding from the anterior nares. The tumor mass was movable and covered by inflamed mucosa but it did not pulsate. There was a chronic excoriation of the skin of the upper lip on the left side.

His nasal septum was displaced to the right, completely blocking that nostril. Examination of the nasopharynx revealed a small amount of adenoid tissue, but no evidence of the tumor. He had a dacryocystitis on the left, apparently due to obstruction and infection of the nasolachrymal duct.

X-ray films revealed atrophy of the left turbinate bones, and marked displacement of the septum to the right with changes in the cribriform region that suggested erosion or possible developmental disturbance. Films taken subsequent to operation revealed an apparent large anterior cribriform dehiscence. The tumor was so solid in appearance it was diagnosed as a fibroma with a possibility of an encephalocele. The diagnosis of encephalocele did not seem probable in spite of the excessive drainage from the left nostril. The tumor had been partially removed when he was 3 months old without any unusual aftereffects and the tumor seemed more solid than cystic.

The difficulties and dangers of surgical interference were discussed with the parents, but they urged that the tumor be removed. The child was becoming a social problem and the risk seemed to them worth taking.

Intranasal operation was performed Dec. 4, 1940, under ether anesthesia. The tumor mass was removed with a tonsil snare and wire. The pedicle was cut cleanly by slow pressure without any traction. Fifteen minutes was taken for tightening the rachet snare, removing a cystic tumor the size of a small lemon. There was very little hemorrhage and no obvious loss of cerebrospinal fluid. After removal of the tumor, the uppor portion of the large pedicle led directly to the anterior cribriform region and could be visualized at the level of the atrophic middle turbinate. The lateral wall of the nose was completely gone and obviously the tumor had occupied the left antrum as well as the nostril.

Dr. Charles Black, pathologist, reported that sections showed a cystlike structure covered externally by nasal mucosa. The internal surface was lined by endothelium for some of its extent but the thick wall of the cyst was composed largely of moderately loose fibromuscular and inflammatory tissue. This wall enclosed a mass of disorganized brain tissue, covered with fragments of what appeared to be leptomeninges. Areas of calcification were found in the white substance of the brain tissue.

Large doses of sulfanilamide were given for two weeks and he made an uneventful recovery. He returned to school and became re-established in his childhood society, reporting every two months for observation.

The drainage from the left nares was now definitely clear and it continued as before. He still had redness of the upper lip from constant coatsleeve and handkerchief mopping, but there were no other complaints.

Nearly a year after operation, he developed a severe headache and a complete cessation of drainage. The mother stated he had not had a cold or sore throat. On Nov. 21, 1941, he was readmitted to Sparrow Hospital with a temperature of 105°. He was in a maniacal semicoma and had a very rigid neck, a positive Kernig, and an obvious meningitis. His spinal fluid was under normal pressure and showed a negative culture, but it was very cloudy and had 12,000 cells per cubic centimeter. Energetic treatment was started using neoprontosil intramuscularly and sodium sulfapyridine intravenously. Within four days his temperature was normal and his mind clear. His spinal fluid cell count gradually returned to normal with ten days of daily drainage.

On Dec. 9, 1941, he was discharged apparently well. Since then, there has been much less watery drainage from the nose, one handkerchief a day being adequate. He does complain of slight vertigo and occasional headaches, but he has returned to school and is keeping up with his class. There is slight recurrence in the upper anterior nares but he has excellent breathing space. He recently had a severe attack of pharyngitis from which he recovered without incident.

The propriety of surgical interference in the case of an encephalomeningocele of this type is debatable. Prior to sulfonamide therapy, nearly all of these patients succumbed to meningitis in early childhood. The boy in Case 1 had weathered several stormy periods of coma which had, apparently, been due to brain irritation. He had, also, survived removal of part of the tumor at the age of 3 months. In his case, the marked social maladjustment, due to the

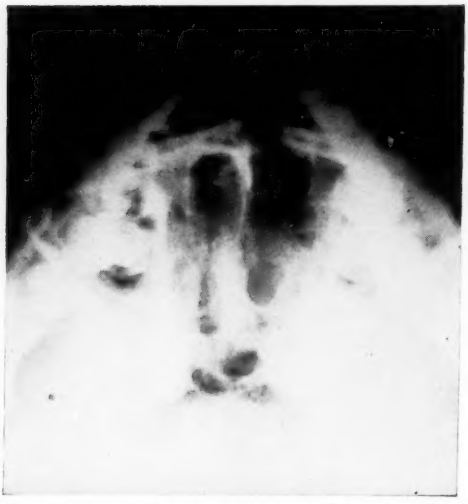


Fig. 6.—Roentgenogram taken after removal of the tumor showing a large trowel-shaped dehiscence of the cribriform plate on the left.

tumor protruding from his nostril, seemed to justify the risk of operation if not the method of approach.

The problem of caring for this patient in the future is disturbing. Probably an intracranial approach to the pedicle should be attempted by a neurosurgeon. On the other hand, he is now very well and happy with only slight drainage from his nose and his family vigorously opposes suggestions of more surgery. There can be no doubt that the threat of meningitis is still present and will be as long as there is any cerebrospinal rhinorrhea. His parents have been instructed to give him sulfadiazine tablets immediately if colds develop, and we hope his olfactory meninges, following the meningitis, may have acquired some immunity to ascending infections.

SUMMARY

The general subject of congenital intranasal cerebral herniations is reviewed.

A case is reported of a 13-year old boy with a large, intranasal encephalomeningocele, operated upon by the intranasal route. The patient did well for a year, then developed meningitis but recovered with chemotherapy.

CONCLUSIONS

Intranasal encephalomeningoceles are uncommon in the literature. However, they are probably occasionally diagnosed as nasal polyps or fibromas.

Some of the cases of meningitis and death that have followed the removal of what seemed to be innocent polyps may well have been mistaken encephalomeningoceles.

If surgical interference seems warranted, the question of the safest approach is debatable, but with the sulfonamides the prognosis is not hopeless.

124 W. Allegan St.

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LI

THE TREATMENT OF DEAFNESS AND TINNITUS AURIUM WITH PROSTIGMIN*†

HARRY SCHLUEDERBERG, M.D.

PHILADELPHIA

The present study was undertaken after the publication by Davis and Rommel of their interesting paper[1] concerning "Treatment of Deafness and Contiguous Nervous Disorders With Prostigmin". Cases of deafness were taken as they presented themselves in the dispensaries of the Episcopal and the University Hospitals. Each case was examined carefully in an effort to classify it correctly as to type of deafness. Before treatment was instituted at least one audiogram was done and in some cases the test was done repeatedly before treatment began. Thereafter, audiograms were done at intervals of about two weeks. Treatment consisted of semi-weekly injections of 1 c.c. of prostigmin methylsulfate 1:2000 hypodermically, and the ingestion of one tablet of prostigmin bromide, each containing 15 mg. of the drug, three times daily after meals on those days when injections were not given. A total of 38 cases were collected. Of these, 20 were treated for three months or longer and 16 for two months or longer. Two cases were treated for less than two months. The latter are included in this study for reasons of interest applying to each which will be mentioned later.

As for the drug, prostigmin methysulfate is a synthetic substance of complicated chemical structure which occurs as a white crystalline powder, quite soluble in water and less so in alcohol. Chemically it resembles physostigmin but its solutions are more stable. Pharmacologically in comparison with physostigmin, prostigmin has a more pronounced effect on peristalsis, a less pronounced myotic effect, and almost complete absence of cardiac by-effects.[2] The reliability of the latter claim may seem to be placed in question by a recent report of 59 patients, three of whom were alleged to

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*From the services of Dr. George M. Coates of the University of Pennsylvania Hospital and of Dr. J. Allan Bertolet of the Episcopal Hospital, Philadelphia.

†Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania, in partial fulfillment of the requirements for the degree of Master of Medical Science for graduate work in otolaryngology.

have developed acute anginoid symptoms.[5] It may be said, however, that in the present series there were no untoward reactions. There was an occasional gastrointestinal disturbance, usually manifested by diarrhea, occasional nausea and, in one case, vomiting, but in this case other medication given concurrently may have been a factor. Two patients reported marked relief from distressing and disabling dysmenorrhea. Another patient, after an overdose of oral tablets, reported profuse perspiration, "quivering" of the muscles of his arms and legs, difficulty in enunciation, diarrhea, unusual mental alertness followed in several hours by fatigue. The drug has been reported upon favorably for the prevention and treatment of post-operative intestinal atony,[3, 4] for the treatment of Ménière's syndrome,[12] and in the diagnosis and treatment of myasthenia gravis.[6] Davis and Rommel suggest it as a new treatment for trigeminal neuralgia but the evidence offered is not very convincing that the drug has any merit for that purpose. An analysis of the 38 cases comprising the present study is offered in an attempt to determine the merit of the claims made for the drug so far as its effects on deafness and tinnitus aurium are concerned.

The patients ranged in age from 16 to 77 years. There were 19 white males and 19 white females. Among the 38 cases there were 20 of perceptive deafness, 8 of conductive deafness, and 10 of mixed deafness. The duration of deafness was from five weeks to 60 years, the average duration of deafness being 12.7 years and the average duration of treatment 2.5 months. Serology was positive in five and negative in 33. Of the 20 patients with perceptive deafness, two showed improvement in hearing and 18 showed no improvement. Tinnitus disappeared in two, was definitely improved in three and doubtfully improved in four, and in seven it showed no improvement. Of the eight patients with conductive deafness, one showed improvement in hearing and seven none. Tinnitus was distinctly improved in two and doubtfully improved in one, showing no improvement in three. Of the 10 with mixed deafness, two showed improvement and eight showed no improvement in hearing. Tinnitus was definitely improved in three, doubtfully improved in two, and showed no improvement in three.

Of the five cases proven to be serologically positive, three were known to be so before accepted for participation in this study while two were found to be positive in the course of routine testing. As mentioned previously, two cases were included in the group which were treated for periods less than two months. Of these, Case 4 is notable because of complete cessation of tinnitus, while Case 17

shows rather remarkable improvement in hearing and tinnitus. Treatment in the first case was discontinued because the patient moved from the city. In the second case, it was deemed advisable to subject the patient to specific treatment when his serologic tests showed strongly positive reactions.

Improvement in hearing was indicated as positive when there was an audiometric reduction of 15 decibels or more in the average for the tonal range of 512, 1024, and 2048 double vibrations. Any improvement of a lesser degree was disregarded as being within normal limits for error. It is true that a number of patients who showed no audiometric improvement whatever, actually appeared to hear better and stated so. However, this is a well-recognized phenomenon. Suggit[7] showed that in a series of cases of otosclerosis, audiometric observations of treated and untreated cases showed improvement in hearing with equal frequency in both groups averaging up to 10 decibels throughout the tonal range. Shambaugh[8] states very truly that "deafened persons are suggestible and stimulated by hope (induced by almost any form of treatment) they begin to pay greater attention and, therefore, seem to hear better."

That there are variables other than the subjective reactions of the patient to be considered in the evaluation of any given case is strikingly borne out by the differences in the conclusions arrived at in this study and in a published report of 56 cases which included 33 cases from this study.[10] Of the cases common to both reports, differences of opinion are expressed as to 11 (33⅓ per cent), a matter which decidedly alters the appearance of conclusions in regard to the value of the drug for the purposes of this study. A careful review of these cases convinces the present writer that the results obtained do not justify the conclusion that "the treatment of deafness and tinnitus aurium with prostigmine is of but little if any value".[10] It seems more reasonable to conclude that in a field where so little of positive benefit is to be offered the patient, any form of treatment that affords any hope of improvement or cure deserves more than peremptory dismissal as the result of observations made on a relatively small group of patients. It is the writer's belief that this drug merits further consideration before it may be said conclusively that it has no place among the myriad of drugs and procedures employed for the purpose.

In evaluating the effect of the drug on tinnitus in this study, the statement of the patient was the criterion for the record. It is easily conceivable that Shambaugh's observation as to the patients being "suggestible and stimulated by hope" may apply here too.

However, there was no doubt about the positive nature of the patient's report in every case where improvement was recorded. In most cases where doubt was expressed, tinnitus was not a very prominent symptom in the beginning. It will be noted that in every case of improvement in hearing in this series, tinnitus too was improved. In this respect, Fowler[9] has stated that "many treatments which are used as so-called cures for deafness appear to improve the hearing because of a coincidental alleviation of the head noises". Five patients showed improvement in hearing, subjectively and objectively and all reported improvement in tinnitus.

It is conjectural as to what may be the mode of action of the drug in improving either tinnitus or hearing. Davis and Rommel[1] offer the embryologic relationship of the aural structures and the facial structures which appear to be favorably affected by the drug in cases of myasthenia gravis as a plausible explanation of its action on structures of like origin. Scal[12] suggests that the beneficial effects of the drug may be due to increased local circulation in the ear. Gleason[11] gives as one explanation for tinnitus, effects produced by distant organs such as teeth, the digestive tract, the nose, etc., on the inferior cervical sympathetic ganglion, stimulation of which by a complicated nervous mechanism brings about dilatation of the arterioles of the cochlea. It is conceivable that a drug which has an antispasmodic effect on the sympathetic system demonstrable in its action on peristalsis, as has prostigmin, might also have such an effect on the sympathetic system at the inferior cervical sympathetic ganglion.

SUMMARY

It may be said, so far as the present study discloses, that of 38 patients treated with prostigmin for deafness and tinnitus aurium:

1. Five, or 14 per cent, showed improvement in hearing.

2. Ten, or 33 per cent, of the 30 patients who complained of tinnitus were improved, with complete cessation of the symptom in two.

3. It is quite possible that the improvement in hearing is due to the improvement in tinnitus.

4. The conclusions drawn in this paper are at variance with those of a previous paper in which the same case reports were part of a larger group.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, Nov. 3, 1941

THE PRESIDENT, DR. JEROME STRAUSS, IN THE CHAIR

The Development of the Olfactory Nerve, the Nervus Terminalis, and the Vomeronasal Nerve in Man*

ANTHONY A. PEARSON, M.D.

(By Invitation)

(This paper appears in full on page 317 of this issue.)

DISCUSSION

DR. WILLIAM F. WINDLE: I knew we would hear a very nice dissertation on the nervus terminalis and the olfactory nerve, because I have been close to Dr. Pearson's work and am familiar with what he has been doing. I know I would be presuming to try to tell you anything more about the embryology of these interesting structures. The adult anatomy, too, has been well covered, and I am left with little other than functional considerations. Naturally I thought you would be interested in the meaning of these structures in terms of adult physiology; but we come to a dilemma here because we must admit we do not know.

This is work on the frontier. Perhaps you do not realize it, but our knowledge of the development of the intrinsic structure in the brain of man is practically nil. The work of Wilhelm His in the last century, summarized in 1904, still stands as the basis of much of our information. So far as our knowledge of the intrinsic

*From the Department of Anatomy, Loyola University School of Medicine, Chicago.

structure of the brain goes, there has been little advance beyond His' study. Only in the last few years has progress begun again. Much of our knowledge of the structure and function of the adult human nervous system depends on working out embryologically the gradually expanding structures as Dr. Pearson has done here. I think investigations of this type, which are careful efforts to explore some of this unknown territory, are of foremost importance. I think he has done a splendid job on this.

It would be speculating to try to say what the function of the nervus terminalis is; or what the meaning of Jacobson's organ is. I cannot help feeling that this nerve is not just a transient thing in the embryo, disappearing in the adult. The suggestion that it may be both afferent and autonomic is reasonable. We now have a picture of the nasal mucosa innervated not simply by two nerves, the olfactory and the trigeminal, but by several others as well. Fibers from the sympathetic reach it and parasympathetic neurons come to it from the facial and sphenopalatine ganglions. Now we see that the vomeronasal and terminal nerves may contribute afferent and efferent fibers. The course of the nervus terminalis into the forward part of the brain, the pre-optic area, and anterior hypothalamus is extremely interesting. Here are located important autonomic centers. The hypothalamus is closely related to the hypophysis and known to be concerned with various sympathetic functions. The nervus terminalis may be an important link in this system. I think we are faced with a challenge to go ahead and see just what the meaning of this nerve is, what it does and what role, directly or indirectly, it plays in the innervation of the nose.

DR. SAMUEL PEARLMAN: It is proper for clinicians to listen to dissertations of this character. The physiology of the nose is to a large extent an unexplored field. A few names stand out. Proetz in our specialty is foremost in his investigations, and his recent text should be read by us all. A neurologist, Elsberg, leads in studies on olfactometry, a field in which, I am afraid, most of us are ignorant.

As I understand it, in man there is a true olfactory system starting in the Schneiderian membrane and ending in the olfactory bulb with its central connections. Another system, the vomeronasal, starts in Jacobson's or the vomeronasal organ, and has its nerves end in a so-called accessory olfactory bulb, better named the tuberculum vomeranosale. In man this system is generally atrophic, but Jacobson's organ can be seen occasionally in the adult. V. Ruysch

showed pictures of it more than 150 years ago, and it is possible at times to insert a probe to a considerable distance.

The relation of the nervus terminalis to Jacobson's organ has been disputed, but it appears to be definitely settled, especially by Herrick, Brookover, and McKibben, that the relationship was that of contiguity and is not functional. There is, further, a ground for speculation as to the function of the nervus terminalis and its central connections, because of its close association with olfactory pathways, but no proof exists today that that function is olfactory.

DR. ROBERT DINOLT: I wish to speak a few words about anatomy and pathology of the olfactory bulb, which in general do not receive sufficient recognition. Anatomic knowledge is highly important for the understanding of the physiology, as Dr. Pearson and Dr. Windle have pointed out. When we inspect sagittal sections of the olfactory bulb we find on its tip a cone-shaped body, described by Brunner in 1923 and called *"Bulbuskoerperchen."* It is separated from the actual end of the bulb by a shallow but clearly marked sulcus. It is no accidental finding, as I have been able to observe it in 20 of 22 examined specimens. It is a body by itself. It contains granular cells and myelin fibers, which are arranged in bundles. These bundles perforate the glomerular layer and the external granular layer of the olfactory bulb, and attach themselves to the inner myelin fibers of the bulb. We have no opinion as to its function and could not trace it to the terminal nerve as has been suggested.

In view of recent publications I wish to draw your attention to some observations regarding the pathology of the olfactory bulb. It has been stated that the presence or lack of the glomerular bodies may give an indication of the atrophy of the olfactory fibers, based on the idea that the mitral cells are never affected by regressive changes. But Brunner has been able to show in 1923 that the mitral cells degenerate even in persons with normal noses. In my material of olfactory bulbs of children and adults we observed two types of degeneration; one with participation of glia cells and one with shrinking and damage to the mitral cells alone. The type with participation of the glia was more often encountered and all stages from increase in satellite cells to neuronophagia and finally coffin formation were seen.

There is no doubt that the number of mitral cells may vary to a great extent within normal limits, as we have seen a great

amount of normal mitral cells even in old individuals, in spite of the presence of regressive changes. That these regressive changes were confined to the mitral cells can be proven by the fact that the ganglion cells of the Bulbuskerne (an accumulation of large cells within the center of the olfactory bulb) never showed any changes. It seems that the mentioned degenerative changes of the mitral cells in the olfactory bulb are the result of a lesion of the olfactory mucosa of the nose, although that cannot be said with certainty, as a number of noses were apparently normal for inspection and we still later encountered degenerative changes of the mitral cells.

DR. WALTER THEOBALD: It is very stimulating to listen to such an excellent and technically clear presentation as this. It would be interesting to have a clinician's viewpoint, and with us is a man who has done considerable work on this subject, Dr. Brunner.

DR. HANS BRUNNER: I wish to congratulate the officers of the society for placing this very interesting subject on the program.

It is interesting to note that the anatomists and pathologists of past decades were mainly interested in the olfactory function of the nose. Thus one finds a large chapter on the olfactory nerve and its central pathways, in the monumental book of Zuckerkandl. Later, when the pathology, symptomatology, and treatment of the paranasal sinuses were worked out, the olfactory nerve was almost forgotten, at least by neurologists. Therefore, I consider it a credit to Proetz that he devoted a chapter of his excellent book to the olfactory nerve. However, if I remember correctly, he did not mention the "physiologic" degeneration of the olfactory bulb which was demonstrated tonight by Dr. Dinolt and which seems to be of considerable interest. The interest in the olfactory nerve has not only a theoretical, but also a practical character. I would remind you of the anosmia following head injuries, the anosmia in meningiomas of the olfactory rim, the olfactory aura in tumors of the mesial part of the temporal lobe; and also of the recent studies of Elsberg concerning the pathologic changes of the olfactory function in cases of brain tumors in general. Considering all these facts, it is obvious that the olfactory nerve deserves a greater interest from the standpoint of the clinician.

DR. ALFRED LEWY: I failed to understand whether the nervus terminalis remains throughout life, or whether it is a vestigial organ like Jacobson's.

DR. ANTHONY A. PEARSON (closing): I want to thank the members of this society for their interest, and particularly Dr. Windle and Dr. Pearlman for their discussions. I am quite familiar with Dr. Pearlman's paper on Jacobson's organ. I had not planned to study Jacobson's nerve (the vomeronasal nerve), but it was necessary in order to understand the nervus terminalis. The two nerves fit together like hand and glove. The accessory olfactory bulb in man has recently been demonstrated. It is small in man when compared with the rabbit where the vomeronasal nerve can be easily dissected and followed to its attachment to the brain. In man the nerve is small and difficult to follow.

I do not think anyone has dissected out the nervus terminalis and demonstrated its course in the nasal septum of adult man. This nerve is quite prominent in the human fetus and infant.

The anterior branches of the nervus terminalis in the nasal septum form a delicate network which resembles the intricate plexuses in the intestine. In the oldest fetuses studied for this report, the glands in the nasal septum had only begun to develop. It may be that some of these fibers are related to these glands.

Gunshot Wounds of Frontal and Temporal Bones

ROBERT HENNER, M.D.

(This paper appears on page 424 of this issue.)

DISCUSSION

DR. SAMUEL PEARLMAN: There is little I can add to this very interesting discussion of bullet wounds of the skull. Rarely seen in private practice, they are occasionally encountered in public hospitals especially of the prison type, one of which Dr. Henner attends. Setting aside the immediate dangers of gunshot wounds of the head, their removal when the patient survives offers frequently unlooked for difficulties. Bullets frequently imbed themselves deeply and firmly in bone, where they are surprisingly well tolerated as a rule. Once located they resist removal stubbornly, and a forceps must be firmly placed beyond the equator of the missile if it is to be loosened. In spite of the fact that they produce often no symptoms beyond their presence, patients frequently wish to have them removed; a psychologic urge, the explanation of which is beyond my

understanding. Bullets inside the cranial vault are properly in the province of the brain surgeon, although I remember, with admiration, the skill and ingenuity with which the late George Boot of this Society successfully attacked many of these problems.

DR. THOMAS C. GALLOWAY: We had a boy at County Hospital who had shot himself in the forehead three days previously, and the bullet made a stellate fracture through the frontal bone and exploded brain substance through the wound. There was contaminated brain tissue exposed in the wound, and it looked hopeless. We coagulated this infected brain tissue with surgical diathermy and it healed up without any trouble.

DR. G. H. MUNDT: The most interesting gunshot case I have seen was a lad whose father collected old guns, and he was shot with a Lüger pistol by another boy. It was a steel-jacket bullet which entered 2.5 or 3 cm. posterior to the outer canthus and exited at the same location on the other side. The only evidence of injury was that one eye was slightly injected. The boy was totally blind. One globe contained a great deal of blood, the other had apparently not been injured. Eventually, when the blood absorbed, one could see the optic nerve evulsed. He was perfectly well except for loss of vision. The other globe was blind from optic atrophy, seen some months later.

DR. JEROME STRAUSS: A patient was in my offiice today who was shot accidentally many years ago and has a bullet in the posterior wall of the left antrum. She says she was shot through the right nostril, because there was bleeding from the right nostril at the time. I have had pictures for a number of years. There is now no sign of where it entered—no hole in the septum, no scar, and no deformity in the nose. She is perfectly all right, though every once in awhile she feels something should be done about it.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, Dec. 1, 1941

THE PRESIDENT, DR. JEROME STRAUSS, IN THE CHAIR

Osteomyelitis of the Superior Maxilla With Recovery Under Treatment With Chemotherapy

ALFRED LEWY, M.D.

Osteomyelitis of the superior maxilla carries a high mortality even under the best surgical management. Therefore, in the case to be described, which was seen very early—so early, in fact, that there could be doubt as to the diagnosis—it was deemed advisable to try chemotherapy without surgery until definite accumulation of pus or necrosis could be determined. There is considerable difference of opinion in surgical circles as to when surgical intervention is indicated in osteomyelitis, but practically all the cases reported are those of the long bones. Ainsworth and Lockhard Smith,* reporting 45 cases in the Brooklyn Medical Hospital, claim far better results by awaiting localization before surgery. In addition, patients received chemotherapy and supportive treatment. Calonna† advocates supportive treatment, then drilling for drainage. Many surgeons, however, hold that incision and drainage should be instituted as soon as the diagnosis can be established, and the earlier the better.

This patient, G. M., a 7-year-old girl, entered the Illinois Eye and Ear Infirmary on June 26, 1940, with an entrance diagnosis of orbital cellulitis, right-sided pansinusitis, following a cold two weeks before. Swelling of the right eye had begun two days previously and was increasing. The left nostril permitted breathing, but the right nostril was completely blocked by swelling, and no landmarks were discernible. Mucopus oozed between the folds of the swollen mucosa. The right eye was red, tender, chemosed; the lids were edematous, the edema extending to the lids of the left eye, but the right eye was not proptosed. There was hard swelling of the right side of the nose and maxilla, extending to the malar bone; this swelling felt like dense bone covered by edema. There

*Med. Times 68:268 (June), 1940.
†J. Kansas Med. Soc., December, 1939.

was no neck rigidity, and Kernig's sign was doubtful. The right drum membrane was grey and without landmarks, and there was a slight serous discharge therefrom but no perforation could be seen.

Blood culture was made but no report was received. The edema overlying the cheek gave the impression of fluctuation but puncture with a cataract knife down to the periosteum found neither pus nor bony dehiscence. The child's temperature was 104°. Washing the right antrum through the natural opening brought out dark fluid blood only. The child was typed for transfusion, but this was unnecessary because of the rapid improvement on sulfathiazole therapy. The blood level was kept at 7.4 for four days and therapy was then discontinued because of the general improvement. The first blood count showed hemoglobin, 77 per cent; RBC, 3,350,000; WBC, 12,200; neutrophiles, 62 per cent.

In addition to chemotherapy administration, the antrum was irrigated through the natural opening, in spite of the absence of landmarks; after the first washing purulent material was returned. Hot dressings were applied and the infra-red lamp used.

The child was discharged in July, 1940, apparently fully recovered. When seen six months later she seemed entirely well and there were no local symptoms in connection with the nose, sinuses, or eye.

The diagnosis in this case may be said to have been not fully established, but the palpable bone swelling on the anterior surface of the maxilla, the extension to the orbital contents, and the finding of dark fluid blood only on first irrigation of the antrum, make this diagnosis very probable even in the absence of a positive blood culture. At any rate, it offers a reasonable suggestion that surgery may be withheld until the diagnosis is fully established. The X-ray showed a dense cloudiness of the antral, ethmoidal and frontal regions, but no bony necrosis could be definitely made out.

DISCUSSION

DR. JOSEPH C. BECK: I saw this patient two days after her admission and I made a diagnosis of a sinus condition with associated orbital cellulitis so often seen with edema. I thought it might be a severe osteoperiostitis, but never thought of its being a true osteomyelitis of the upper jaw. Of course we will never know, because as Dr. Lewy said, the treatment was successful. Another important fact in the successful outcome of the case is that the two excellent young men who were residents at the Infirmary were the

best men in sticking to a case I have ever seen. They kept a close watch on the saturation point of the blood, as well as the blood count.

One point was not emphasized sufficiently; one ear was mentioned as acutely discharging, but on looking through the record I find there was a bilateral otitis media acuta. There was at one time a threatened mastoid condition. No operation was performed, however, and both ears are now well. When I saw her first I advocated early opening of the fluctuating subperiosteal abscess; I thought drainage would prevent spread of the infection.

The outcome in this case gives us the hopeful thought that by means of the treatment so courageously followed by Dr. Lewy we can avoid operation in many cases. Dr. Lewy is to be congratulated on adhering to this treatment as he did, and I feel he deserves much credit; or shall we say both he and the patient were fortunate if, as he says, the case was one of osteomyelitis.

Dr. Howard C. Ballenger: I would like to ask Dr. Lewy what organism was recovered. The few cases of osteomyelitis of the superior maxilla that I have seen have been infants, in which fistulas developed, or following injuries with fracture of the superior maxilla. I have suspected a localized osteomyelitic process in a few cases in adults following an apical root abscess at some distance from the floor of the maxillary sinus in which erosion into the sinus finally occurs.

Dr. Sherman Shapiro: I should like to place this case on record. The patient, a young man, in the summer of 1938 sustained a crushing injury of the left superior maxilla through the agency of a large meat hook. When I saw him about four months after the accident, he had a discharging fistula at the orbital margin and tearing. Irrigation through the natural opening on this side was followed by a good deal of pus, and X-ray showed marked involvement of the antrum with some involvement of the orbital rim. I operated by the Caldwell-Luc method in an attempt to get drainage through the nose, enlarged the external fistula and connected it with the antrum. The nasoantral bony wall was found to be gone completely and all we had was mucous membrane.

The organism in this case was Strep. hemolyticus beta in pure culture. In postoperative treatment sulfanilamide was of no value, either in powder form locally or internally. X-ray some months later showed that the osteomyelitic process was progressing. At the second operation I took about half the orbital floor and left it open

again externally. It was kept that way for a year, and at the end of that time X-ray showed that a lot of repair had taken place and the antrum condition seemed to be clearing up, so I did a Toti-Mosher tear sac operation to insure drainage into the nose from the tear sac. I closed the external fistula, and the boy has been well since last April.

This boy was under treatment for more than two years. Nothing seemed efficacious until drainage was established. There is still a little tearing in cold weather, but otherwise he is well. The entire bony wall was gone at the time of operation, but at the time of his discharge it had regenerated. I took out large sequestra at both operations, and am now showing the final result.

Dr. Greenwood asked about the teeth. No culture of the teeth was obtained during the course of the treatment. All the devitalized teeth were extracted in the attempt to hasten recovery.

DR. THOMAS C. GALLOWAY: I think we ought to understand what we are talking about when we discuss osteomyelitis of the maxilla. The maxilla has very little medullary structure, and probably can have what Dr. Beck called osteoperiostitis, but not an osteomyelitis. There are various conditions which lead to this. We have all seen cases which, in spite of anything that could be done, progressed to involvement of other cranial bones and death.

Dr. Lewy did not report the organism found, which is important. A 21-year-old boy, the son of one of the best known pathologists in town, after an antrum window operation developed such advancing infection with a fair amount of sepsis. There was no response to chemotherapy. We obtained a culture of anaerobic streptococci of the type Melanie has shown to be so important. It is difficult to stop, but responds to zinc peroxide. I made an incision through the canine fossa and over the inferior turbinate, and injected this twice a day. The boy made a startling recovery. I shall soon report about six similar cases. This organism is very small and grows very slowly, and routine examination will not show it. It seems to me the presence of an organism such as this explains some of these cases. Zinc peroxide is so specific that perhaps we have a remedy of considerable value for these cases.

DR. ALFRED LEWY (closing): I have a great advantage over Dr. Beck. As a special instructor for residents and interns he frequently sees cases before I do. He says I should operate and I think I should not, and vice versa, but we really seldom disagree.

In reply to Dr. Ballenger concerning the origin of this infection; we paid no attention to the ethmoid, which X-ray showed to be as cloudy as the antrum. I have seen a number of cases of orbital cellulitis secondary to antrum infection. We were safe here in assuming that this was the original focus of infection. I first washed out bloody fluid and then pus from the antrum.

Dr. Shapiro's case is, of course, a well-developed osteomyelitis; my case may have been an osteoperiostitis. I do not believe we could make a distinction. Nevertheless, I believe the course shows it is possible in some cases to avert surgical osteomyelitis.

About two years ago Dr. Galloway told me of the success of the zinc peroxide treatment in presumably anaerobic infections. I had heard of its use for spreading cellulitis of the abdominal wall and was about to try it on a case of gangrenous osteomyelitis of the nose and accessory nasal sinuses which I reported several years ago, but unfortunately that day the patient died of a hemorrhage of the internal maxillary artery. However, I did pass Dr. Galloway's suggestion on to a colleague who had a similar case.

Observations on Acoustic Trauma in War and in Peace

HENRY B. PERLMAN, M.D.

(Abstract)

In reviewing the growth of our knowledge of acoustic trauma, the importance of the audiometer in detecting early degrees of this condition was pointed out. Many examples were shown of the hearing curve characterizing this early lesion, an abrupt high tone loss with or without a depression maximum at about 4096 cycles. Experimental data on the relation between the frequency of the traumatizing tone and the resultant threshold depression were presented. This experiment showed that tested at octave frequencies the maximum depression in threshold was an octave above the frequency of the fatiguing tone and that high frequency sounds were more traumatizing than low frequency sounds of an equal intensity. No uniform abrupt high tone loss or 4096 dip was found in these subjects traumatized with pure tones.

The military aspects of acoustic trauma were then presented with a special consideration of the shock pulse. This is a peculiar

pressure phenomenon produced by bombing, a gun blast and by the nose and base of the moving missle. Quantitative data on the pressures developed by these shock pulses and their duration and propagation were presented and integrated with our knowledge of the latent period of the protective middle ear muscle reflex and the manner in which the cochlear and organ works. This explained the reason for the acoustic sensations produced and for the trauma to the perceptive apparatus. The discussion was integrated with observations on ear injuries due to the shock pulses from bombs made in Spain during the Civil War.

DISCUSSION

DR. C. C. BUNCH: This study by Dr. Perlman, together with the one by him which appeared in the September *Archives of Otolaryngology*, has been of great interest to me because in several respects they tend to verify the observations made in a paper which I read before the American Otological Society in 1937. For that study we searched for material for two years, carefully scrutinizing each record of this type, before it was decided that we were on the right track. When audiograms of this peculiar type were discovered, we questioned the patients very minutely about the possibility of acoustic trauma. Whenever anyone about the clinic was known to have been exposed to loud sounds, we tested their hearing to see if there was any evidence of hearing loss. Later on, we made it a practice to inquire of all men over 35 years of age who came to the clinic, if they had served in the army or navy, and if a positive answer was secured, this type of loss was found in a surprisingly large percentage.

The study I presented was less than half completed, for we were unable to establish by experiment a definite cause-and-effect relationship. Dr. Perlman has in a limited way attempted to do this. Frankly, I was afraid to expose anyone to a very loud sound for a long period of time for fear the exposure might result in permanent hearing loss. I have had permanent loss of this type in both ears for more than twenty years. The loss was due, I think, to a single exposure. I did not wish to lay myself liable for damages.

It has been stated frequently that the audiometer gives no information which cannot be secured by the older tuning fork tests. In this case, however, it appears that while tuning forks in the hands of our otological teachers did point out the general character of the loss from acoustic trauma, the audiogram pictures it much more definitely than was possible with tuning fork tests. Otherwise I

think Politzer and the other early investigators would have defined it more definitely. I believe Dr. Perlman is correct when he states that the tonal dips may appear at other frequencies than 4096 cycles. In one of my ears, the deepest part of the dip comes at 6100 cycles and in the other at 6300 cycles, although both ears show considerable loss at 4096 cycles. In my own case the dips were located by means of a pitch range audiometer. For this reason there can be scarcely any doubt that with an audiometer which produces only the octave tones we are almost certain to miss the dips which come at other than the octave frequencies.

This first lantern slide shows a dip in both ears at 1448 cycles which would have been missed if only the octaves had been tested. In this case the loss was either congenital or acquired before speech habits were formed.

We suspect also that high tone losses of this peculiar type may in rare cases come from other causes than acoustic trauma. Dr. Perlman in his first study mentioned intoxication, syphilis, trauma to the head and retinitis pigmentosa. I have found them only in cases which I thought were of congenital origin. The second slide shows two records which were almost identical. One was, I think, of traumatic origin and the other congenital. The third slide shows the records of a mother and five daughters, three of whom show very marked loss for high tones with normal hearing for low tones. We think the loss in these three was congenital or acquired before speech habits were formed, for all three had similar speech defects.

Dr. Sherman Shapiro: There are two points connected with this paper that should be brought out. In some circles abroad differentiation is made between explosion trauma and acoustic trauma. By explosion trauma is meant that which occurs mostly in wartime, and occasionally in peacetime as a result of extensive detonation. By acoustic trauma is meant the chronic type illustrated by Dr. Perlman. I think the differentiation should be made. From the scientific standpoint we know, by autopsy, that a bomb blast causes multiple hemorrhages in lungs, liver and other internal organs as well as the inner ear. This factor can certainly account for war deafness as much as the noise of the blast. From a practical standpoint I think the differentiation should be made because in the sort of thing we see in peacetime, from exposure to noice such as in boiler factories, the deafness is different from the result of explosives in war.

From following Dr. Perlman's experiments, is it not correct to say that the upper tones are more vulnerable and that any trauma will affect this part of the scale first?

DR. ALFRED LEWY: In considering acoustic trauma, one factor is possible, that of vibrations transmitted through the bony skeleton of the body, as in certain industries. That would also include aviators who are exposed to such tremendous vibratory impulses.

DR. HENRY B. PERLMAN (closing): Dr. Shapiro spoke about the sensitivity of the cochlea to high frequencies, as shown by these audiograms. The importance of high frequencies in industrial noise is only part of the picture; the rest is concerned with the brief sound pulses about which we know very little and which cannot be analyzed as ordinary sounds. These shock pulses are particularly important in acoustic trauma, because of their intensity and brief duration (less than the latent period of the middle ear muscle contraction). I think the difference between explosion trauma and acoustic trauma are differences only in the degree of injury to the perceptive apparatus and not in its duration or type. Both the shock pulse of an explosion and the sound wave of a noise produce a localized irritation of the cochlea.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, Jan. 5, 1942

THE PRESIDENT, DR. JEROME STRAUSS, IN THE CHAIR

Air Pressures in the Nose and Maxillary Sinus Under Normal Conditions and in Disease

DELBERT K. JUDD, M.D.

(Abstract)

A preliminary report was presented on investigations of fundamental problems related to the etiology of sinus infection and the principles underlying methods of therapy. Some previous experimental studies were repeated and new investigations carried out. The investigations include:

1. Comparisons of pressures in the nose and paranasal sinuses during normal physiologic processes such as quiet respiration, blowing and sniffing, under both normal conditions and in disease.

2. Observations on the effect upon pressures within the sinuses of (a) mass suction, (b) irrigation, and (c) combined suction and irrigation.

Under normal conditions pressure equilibrium is maintained at all times between the nasal and sinus cavities. Blowing causes greatly increased pressure in both the nose and the sinuses. During the act of sniffing there is marked but equal decrease in nasal and sinus pressure. When partial obstruction of the ostium from any cause is present a time lag occurs before the peak pressure in the sinus equals pressure change in the nasal fossa. The new pressure within the sinus tends to be maintained until exaggerated opposite pressures are produced in the nose. The time lag in establishing pressure equilibrium is a direct indication of the amount of obstruction at the ostium, and of impediment to the efficiency of the ciliary stream. With complete obstruction of the ostium there will be no alteration of the pressure existing in the sinuses regardless of varying pressures which may be set up within the nose.

The relationship of sinus and nasal pressure changes under different pathologic conditions was demonstrated by means of curves projected on the screen.

Observations on sinus pressures were made also under the following conditions:

1. Simple mass suction: The observations showed the method to be extremely inefficient in removing secretions from sinus cavities except when the sinus is partially filled with air and the fluid overlies the ostium. Under such conditions some fluid is removed but only a fraction of the sinus content. (These observations do not include interrupted negative pressure which is the basis of displacement therapy.)

2. Simple irrigation (irrigation without addition of suction): The irrigation is accompanied by positive pressure within the nose and (if the ostium is patent) also in the sinuses. Secretions may be removed from the nasal cavity but not from sinuses.

3. Irrigation combined with suction: This is the principle used by nasal siphons and suction irrigators. On analysis this method is a combination of mass suction and an irrigating stream. The effect on the sinuses is precisely that of mass suction and is open to the same limitations.

DISCUSSION

DR. O. E. VAN ALYEA: I like the way Dr. Judd has presented this subject and brought us up to date on some of the physiologic concepts which should guide us at the present time in our treatment of sinus disease. Some years ago O'Malley, of London, brought out the fact that there was a negative and a positive presure change in sinus cavities, based upon inspiration and expiration. He carried the theory to practical application. He felt that on inspiration we obtained a definite removal of secretions from the neighborhood of the ostia of the sinuses due to the pressure changes. It seems to me that this pulling effect is similar to that produced by a passing streamliner train; the suction as it passes might take your hat off. I have always felt that secretions carried to the sinus ostia by the cilia and retained by a small ostium are quite often pulled through the ostium by an inspiratory blast. Hilding emphasized the involvement of ciliary action based on these changes. He has shown that in rabbits, when the anterior naris is sewed up, there is interference with ciliary action. He sewed up one anterior naris of a rabbit, left it for three weeks, and found after removal of the

sutures that the mucosa of both nasal cavities showed fragmentation of the cilia, and some destruction of the mucous membranes.

In another experiment, McMurray instilled iodized oil into both antra of a human subject and blocked off one naris. X-rays at intervals showed the oil unmoved for seventeen hours on the occluded side, while on the open side oil had passed out of the ostium and reached the floor of the nose in five hours. After removal of the occlusion, the oil on that side started to move at the same rate of speed and also reached the floor in five hours. Negative pressure caused by inspiration is shown by this experiment to be of vast importance in the evacuation of heavy material from the sinuses.

One of the important things Dr. Judd has given us is his conclusion regarding mass suction. I think most of us feel that clinically mass suction is of very little value in promotion of drainage of the sinuses and the procedure is often harmful.

Anything we can do to improve our understanding of nasal physiology is certainly to be commended. This work of Dr. Judd's is instructive and timely and, consequently, highly commendatory.

Dr. Alfred Lewy: The mechanical contrivance known as an injector has been used by engineers to produce a flow from adjoining cavities through a pipe, simply by passing a jet of steam or air through a small pipe past an ostium corresponding to the nasal sinuses. That is what we do when we blow the nose.

There is one point in mass suction which seemed to be lost sight of by Proetz and others. It may not such a discharge from the sinuses unless the discharge lies at the opening. There is the question of the therapeutic effect of hyperemia induced by suction. This is a good idea under certain circumstances, though probably contraindicated in the acute stages.

Dr. Thomas C. Galloway: This work seems to have been carefully done, and the conclusions should be valid. I must admit I did not quite follow all Dr. Judd's logic and shall be interested in studying the written article. I still think suction may be very valuable, but it must be used logically and gently, and with the visualization of what you are going to do. If one takes a model and carefully turns the ostium into the direction in which the secretion will overlie it, with air above that, one can certainly empty even thick secretion if the ostium is in the proper position. Of course, suction does not work in the absence of air in a sinus. It is obvious

that it should not be used to the extreme of producing hyperemia or rupturing vessels.

What happens to the blood vessels with suction? Plus and minus pressure should increase the circulation of the blood and aid in proper nutrition of the cilia and cells. Also, increase of negative pressure in the sinuses, or absorption of air by blocking the ostium might lead to edema and possibly cupping action on the capillaries producing marked increase and possibly hemorrhage.

Dr. Walter Theobald: Years ago it was the opinion that the maxillary sinus most frequently involved was the inaccessible one. Some years ago I felt we could disprove that theory because we were able to get into so many antra either by natural or accessory openings, and in a series of 100 cases I determined that 52 per cent had accessory openings. I want to bring this to your attention. The beautiful explanation Dr. Judd gives us shows that the sinus most frequently involved is the one easily accessible; therefore, the one with an accessory as well as a natural opening is most frequently involved. If you will follow some cases you will note that the cannula frequently slips into an accessory opening when you think you may be in the natural opening.

Dr. G. H. Mundt: I have often noted the frequency with which the maxillary sinus is involved on the side of the concavity of the septum, and I think this theory of Dr. Judd's does much to explain that. I do not know whether anyone will agree with me, but undoubtedly the pressure is an important factor in producing more frequent involvement on the side of greatest concavity.

Dr. Delbert K. Judd (closing): In the time allotted it would be confusing and ineffectual to attempt consideration of the apparatus or mass of observations connected with the work done. The examples shown by the curves and charts were thought to be sufficient for illustrating the purpose and conclusions of the investigation.

Dr. Van Alyea compared the nasal air stream to a streamlined train, to illustrate a process frequently experienced when a large mass of mucopus has been partly extruded through the ostium so that a portion of it extends into the path of the air stream. Forceful efforts of sniffing and blowing hurl a swift current of air against the mass, moving it backward and forward and finally pulling or dislodging the entire mass from the sinus. However, this mechanism comes into play only when the cilia have expelled a sufficient

amount of the mass through the ostium to be caught in the air stream.

The injector principle spoken of by Dr. Lewy, as it exists at the ostium, is comparable to mass suction and could be effective only to that extent.

Perhaps, as Dr. Lewy says, the hyperemia produced by massive suction is an important therapeutic measure. I respect the great amount of clinical experience upon which Dr. Lewy bases his statement and I have no reason to disagree. I would only emphasize the fact that mass suction, being difficult to control, may exceed the negative pressure which can be tolerated by nasal mucous membrane. According to Proetz, negative pressures in excess of one-fifth of an atmosphere may cause hemorrhage which is not always evident since it may remain submucosal. If infection of the hematoma occurs serious complications are possible.

In answer to Dr. Galloway, I agree that a small amount of secretion can be withdrawn from a sinus by mass suction provided the fluid overlies the ostium when the negative pressure is applied. The amount of discharge recovered, however, is only equal to the volume by which the air in the sinus is increased by the decreased pressure. This is an application of Boyle's law.

Since nasal mucous membrane will tolerate a decrease of only one-fifth of an atmosphere of pressure, the amount of fluid which can be obtained is certainly limited. Air must be present in the sinus before this mechanism will work. No secretion can be withdrawn from a completely filled sinus by mass suction. Other factors which hinder this type of treatment are secretions of high viscosity and partially and completely blocked ostia.

I would like to add to Dr. Theobald's discussion of the accessory sinus ostia, a fact not generally known. The studies of Proetz negate the supposition that an accessory ostium will permit a through-and-through flow of air. Pressures in the nose act upon both openings equally and simultaneously, thus maintaining equilibrium between them.

Labyrinthine Dropsy, Inner Ear Deafness, and Ménière's Syndrome

JOHN R. LINDSAY, M.D.

(Abstract)

The true Ménière's syndrome or disease may be divided into three stages or groups:

1. The early stage when only vertigo occurs, sometimes called "pseudo-Ménière's." Vertigo is frequently postural. Conditions to be differentiated include toxic disturbances of vestibular system, early multiple sclerosis and early brain tumor. Cases of this type may recover completely or progress into the second group and develop the true Ménière's syndrome.

2. True Ménière's syndrome: Attacks of vertigo vary in frequency, severity and duration. They are usually preceded by a sense of increasing fullness, or deafness, or tinnitus, or all combined, culminating in an attack of vertigo. The vertigo attacks occur over a period of a few years, then subside. The deafness persists and usually increases.

Characteristics of the deafness are: Wide fluctuations in the threshold, especially for the lower frequencies. Variations of up to 40 db. in the threshold may occur depending on whether the test is made preceding or following an attack.

Bone conduction remains good in comparison to that occurring in nerve degeneration. The Rinne is shortened positive. The upper tone limits are well preserved. Recruitment is usually partial. Diplacusis or a disturbance in the tone quality is an intermittent accompaniment of most cases.

3. As the vertigo attacks decrease and disappear the remaining disturbance of function is deafness. The hearing loss progresses and fluctuations decrease. In this stage the previous occurrence of vertigo may be overlooked unless specific inquiries are made. Possibly also vertigo in the early stages may may be only slight.

Case histories and hearing records were demonstrated to illustrate the three stages of this disease.

The discovery of labyrinthine dropsy in Ménière's disease was made by Hallpike and Cairns in 1938 and subsequent reports were made by Hallpike and Wright (one case), Yamakawa (one case), and Rollin (two cases).

Rollin reported three patients with labyrinthine dropsy, two of whom were only known to have deafness, and one without available history.

A case corresponding to Rollin's, of bilateral labyrinthine dropsy with advanced bilateral deafness, was presented. The pathology has been described in detail in an article by the author in the *Archives of Otolaryngology*.

The pathologic findings in these ears furnished new and strong evidence to explain the characteristic disturbances of function produced by this disease. On the basis of the evidence it is probable that the auditory disturbances are due not to involvement of the nerve mechanism but to a dampening effect on the fluid movement within the cochlea produced by the distortion of the membranous labyrinth. This explains the unusual characteristics of the auditory disturbances in Ménière's disease.

An explanation for disturbances of equilibrium is also given by the existence of herniations of the utricle into the semicircular canals, both at the small end and at the ampulla, where a dislodging and distortion of the ampullary wall must provide mechanical interference with the normal movement of the cupula within, thereby disrupting equilibrium.

While these findings present a new and reasonable explanation for the disturbance of function in this disease, the etiology remains obscure.

DISCUSSION

Dr. William E. Grove: This paper constitutes a magnificent contribution to the whole question of the pathology of the Ménière complex. No satisfactory explanation or description of the pathologic background of this condition appeared following Ménière's original description in 1867, until Hallpike described the temporal bone pathology in two individuals who died following intracranial section of the auditory nerve. The pathology he described was very similar to that shown here. Hallpike advanced the hypothesis that the attacks of vertigo were initiated by what he called "bouts of labyrinthine asphyxia" brought about by rapid rises of fluid pressure by relatively small volume increases of endolymph. His contention was that only after enormous dilatation of the endolymphatic cavities had taken place would the relatively small increases in endolymph volume operate to produce the typical vertiginous attacks.

In the typical case of the Ménière symptom complex we have the association of vertigo, deafness and tinnitus. The vertigo comes on in typical attacks, the deafness is usually progressive, and at some time or other tinnitus is present. Furthermore, there are long periods of remission in some cases; so long, in fact, that the original vertiginous attacks may be all but forgotten. Lindsay's case report fails to mention any history of vestibular symptoms, but these may have been evanescent and forgotten. With herniation of the endolymphatic wall of the utricle into the ampullated end of the semicircular canals noted in the pathologic specimens, it is rather inconceivable that this individual should never have had any vestibular symptoms. It may be true, however, that the increase in endolymphatic pressure and the effects exerted thereby upon the sensory epithelium may have been so slow and gradual that vertiginous attacks were not set up in this individual. It may also be true that the sensory epithelium of the vestibule and semicircular canals may be more resistant to prolonged but gradually increasing pressure than that of the organ of Corti. Crowe also called attention to the fact that some of the cases of gradually increasing deafness may, in fact, be atypical cases of Ménière's disease.

Whether all cases of the Ménière complex would present the same pathologic picture which Hallpike and Lindsay have described is hard to say. As Lindsay points out, patients do not die of this condition and the temporal bones are not often obtained for pathologic examination.

That hydrops of the labyrinth might explain the symptoms of Ménière's disease is apparent from the previous observations of Dederding, Furstenberg, Crowe and Hallpike. Whether this hydrops is due to disturbance of water metabolism as supposed by Dederding, to disturbed sodium metabolism as propounded by Furstenberg, or to some other as yet unknown etiologic factor, is not clear.

However, it is erroneous to say that dropsy of the labyrinth will explain the symptoms in all cases, for if such were the fact the symptoms would disappear after intracranial section of the auditory nerve or total destruction of the sensory epithelium as practiced by Mollison, Berggren, Putman and others. The symptoms of vertigo and tinnitus do not always completely disappear after these procedures. It is my contention, therefore, that whatever etiologic factor is producing the hydrops probably also produces a "water-logging" of the central cochlear and particularly vestibular nuclei. There is no proof of this except by deduction.

This work of Lindsay is extremely important in corroborating the findings of Hallpike and others, and it is to be hoped that more temporal bones from Ménière patients may become available for pathologic study.

Dr. George Shambaugh, Jr.: This very interesting and instructive paper is a real contribution to our knowledge of Ménière's syndrome. Dr. Lindsay's observations are almost in complete accord with those I made in 1935 and 1936 on diplacucis and fluctuating deafness in this condition. He did not mention that the deafness is a low tone nerve deafness, but the audiograms on his cases show that the tones of lower pitch are involved as much as or more than the tones of high pitch, contrary to the usual nerve deafness where the high tones are involved more than the low ones.

As I pointed out in 1936, I believe there is a syndrome of low tone fluctuating nerve deafness with diplacucis, where the prognosis for hearing is relatively good if we can find an etiologic factor. Food allergy is a factor in many cases. I have a series of cases where elimination of food allergy has resulted in marked improvement in hearing as well as in vertigo. It seems to me that allergy may explain the hydrops of the labyrinth better than anything else. Allergy causes a localized edema with an outpouring of fluid into the tissues, and the histologic findings in the cochlea, it seems to me, could be explained on this basis.

Dr. John R. Lindsay (closing): I showed this slide partly for the reason that this individual showed low tone deafness with symmetrical and normal upper tone limits, positive Rinne, and relatively good bone conduction which fluctuated depending on the air conduction. That is what Mygind and Dederding believe to be a typical picture of this disease. The two cases I showed previously had greater loss in the high tones. I think this is probably a true picture of Ménière's disease. This patient was under care for one year with attacks of vertigo before he developed auditory disturbance.

Regarding the explanation of low tone deafness—that is the reason I pointed out that the nerve apparatus, so far as we can tell by histologic methods, appears to be in good condition. Therefore, I am inclined to think that Mygind and Dederding are right when they say this is due to a disturbance of conduction in the fluid system. They did not know there was a labyrinth hydrops, but I think this bears out their conception that it is an inner ear conduction deafness rather than nerve deafness. I think this conception better explains the fluctuations. When fluid is increased you get

greater deafness, and vice versa. It seems to me the explanation is more reasonable on that basis than to assume you could get such wide fluctuations week after week on the basis of nerve degeneration. I have tried inflating the tube repeatedly when patients felt that an attack was coming on, but could get no change whatever.

Of course allergy must be considered, but when we consider the great number of patients with allergy and the infrequency of Ménière's disease, the percentage where the two appear related is extremely small.

On that basis we have been trying to produce vertigo in animals. We have been giving animals allergic reactions in the middle ear, but so far have not been able to get a nystagmus. That work is not finished yet. This case was put in to illustrate one case belonging in the pseudo-Ménière group for a year, then developing the complete Ménière's syndrome.

I want to thank Dr. Grove for his discussion. As to the etiology of the functional disturbance, Hallpike's explanation was on the basis of increased pressure. That explanation never was satisfactory, and as Crowe pointed out, the labyrinthine fluids are subjected to enormous pressures in certain central nervous system diseases without deafness or vertigo. It is impossible to get pressure in the endolymph much greater than the perilymph because of the extreme thinness and nonelasticity of the membranes, as long as any perilymph remains in the labyrinth.

Observations on the Acoustic Movements of the Human Sound Conduction Apparatus

H. G. Kobrak, M.D.

(Abstract of Motion Picture Demonstration)

Direct observation of the conducting mechanism during acoustic stimulation is very difficult in man for a number of reasons. The anatomic structures are minute and not readily accessible. The amplitudes of the movements are small and, finally, the vibrations so fast that the human eye cannot record them. A number of technical improvements were made to overcome these obstacles.

By using an appropriate optical system all important anatomic structures can be sufficiently enlarged. Mirrors on the vibrating

parts provided a suitable means for objective observations. In this demonstration the value of the moving camera as a recording apparatus was demonstrated. The film begins with a demonstration of the experimental setup. It shows a machine which produces slow air pressure changes of sinus form. These pressure changes can be considered as tones below the lower tone limit. On fresh cadaver specimens these slow movements of ear drum, ossicular chain, stapes and round window membrane are shown in the movie. Acoustic vibrations are next seen. Under both ordinary 16 frame exposure and under "slow motion" photography acoustic movements of the ossicles are demonstrated on cadaver specimens.

The next development of the method was the use of stroboscopic light (120 interruptions per second). Various tones near 120 cycles are directed into the outer ear. The clear visibility of the amplitude of the ossicles is striking as shown in the movie.

The following part of the film deals with the acoustic stapedius reflex in patients with an ear drum perforation and in a radical mastoid cavity. The tendon of the stapedius muscle and head of stapes are seen.

The last part of the movie shows the ear drum of a normal person. A tone is directed into the outer ear. No movements of the membrana tympani can be seen. If, however, stroboscopic illumination is substituted for ordinary light, definite movements of the membrana tympani are demonstrable. With this method it has been possible for the first time to see directly the acoustic oscillations of the ear drum.

Abstracts of Current Articles

NOSE

Nasal Botryomycosis. (Botriomicose nasal.)

Prudente de Aquino, F., Rev. Brasil. de Oto-Rino-Laring., 9:9 (Jan.-Feb), 1941.

The present work deals with an anatomopathological finding of nasal botryomycosis. Description: vegetative and pediculated tumor on the head of the middle turbinate bone, in the right nasal passage. The chief complaints consisted of crisis of sneezing, nasal obstruction and nosebleed. Ample biopsy and removal of tumor, which was as big as a bean, were performed. Bleeding tumor, local cauterization with 3 per cent chromic acid. The histopathological examination, at the São Paulo Faculty of Medicine, established the diagnosis of nasal botryomycosis. The histopathological features of the tumor are described as well as its different names (botryomycosis, pyogenic or telangiectatic granuloma, etc.) The clinical study of the morbid entity is made, and the differential diagnosis with angiomas, fibroangiomas, and equine botryomycosis (castration wound) is made. Anatomopathological considerations about the present case. Discussion about its frequency in dermatology and the rarity of its manifestations on the mucous membranes, especially the nasal one. The treatment and different adopted measures were: galvanocautery, chemical cauterization, etc.

AUTHOR.

Nasopharyngeo Fibroma. (Fibroma naso faringeo.)

Regules, P. (Montevideo), An. de Oto-Rino-Laring. d. Uruguay 10:11, 1940.

The author presents the case history. The fibroma was rather large, hemorrhagic, and there was a large implantation in the lateral choanae. Operative removal was effected after the patient was prepared for the procedure with transfusions and sulfonamide therapy because of a septicemia developed while awaiting surgery. The operative method is described, with emphasis on the importance of ligating the external carotids for better control of the bleeding. Radiographs are shown, as well as pictures of the specimen and microscopic sections.

HERDENER.

Some Considerations on Surgery in the Allergic Rhinopathies. (Consideraciones sobre la conducta quirurgica en las rinopatias alergicas.)

Cortes, J. S. (Guadalajara), An. de Soc. Mex. de Oftalmol. y Oto-Rino-Laring. 16:20 (Jan.-Feb.), 1941.

The author condemns the use of nasal surgery and resections following what he refers to as *a priori* diagnosis of vasomotor rhinitis, stressing the necessity of proper differential diagnosis prior to surgery. Periodic coryza, aperiodic coryza and perennial coryza with their subdivisions and classifications are discussed in their symptomatology and various diagnostic points. Two patients who had undergone unnecessary and traumatizing surgery are cited as examples. The author does, however, point out the necessity of surgical intervention in appropriate cases.

HERDENER.

Some Questions Concerning the Allergic Reactions of the Nasal and Paranasal Mucosa. (Algunas cuestiones sobre las reacciones hyperergicas de las mucosas nasales y paranasales.)

Recciteli, E. (Buenos Aires), Rev. Argent. de Oto-Rino-Laring. p. 170 (May-June), 1940.

In this paper the author considers several factors concerning hyperergic reactions of nasal and paranasal mucous membranes, which he finds interesting in the interpretation of the pathologic etiology of the same.

These reactions are found to be more prevalent in the spring. The organic and mineral metabolic changes in the body at this season, together with other meteorologic changes, contribute in interpreting the physiopathology of the observed changes. The fact that the autonomic system is more liable directly and indirectly to the seasonal variations may explain in susceptible individuals the manifestations of known symptoms.

Various known sensitivity tests are discussed as well as the place this condition could take in the general picture of hypersensitivity. Hereditary factors and the reactions of the nervous system to the above phenomena are included. The hormonal effects and the seasonal changes in the body as they concerned these reactions received the greatest space.

HERDENER.

The Next Step in the Etiopathogenesis of Ozena. (Proximo paso en lar investigacion de la eti-patogenia del ocena.)

Tello, A., Rev. de Otorrinolaring. 1:43 (Dec.), 1941.

The author says that any substance intradermically injected modifies, under certain circumstances, the symptomatology of ozena and neuralgias. Asthma, ozena, neuralgias and rheumatism are to be met in families with allergic background, the manifestations of which assume a reaction to unspecific allergens that varies according to the kind of endocrine, neuro-, or vegetative constitution. As ozena is to be found among such manifestations of allergic constitutions, it is only natural that its etiology should be looked for in that field. Ozena might be thought of as a local anaphylactic phenomenon; just like the Arthus phenomenon.

AUTHOR.

Rhinoscleroma in Chile. (El rinoscleroma en Chile.)

Labra, L. L. (Santiago), Rev. Brasil. de Oto-Rino-Laring. 8:533 (Nov.-Dec.), 1940.

The article is written with a view of stimulating his colleagues into helping to uncover focus areas of scleroderma. First case diagnosed in Chile was in 1896. The author enumerates cases reported in Chilean literature, and discusses two of his own four cases. One of these had been masked for year by an intermittently treated syphilis. Several photomicrographs demonstrating Mikulicz cells, corpuscles of Russel, and foci of the bacilli of Frisch are added to complete the presentations of the two cases.

Generally a discussion of the characteristics of the disease is presented with emphasis on the necessity of guarding against confusing with ozena in the early stages, and differentiating in the late stages from epithelioma, sarcoma, syphilis, and tuberculosis. Therapeutic methods found to be of most value were curretting and roentgenotherapy. Definite foci of the disease are known to exist in Guatemala, Colombia, and Salvador, while those previously reported in Brasil were definitely shown to be erroneous diagnoses. The question is finally brought up with regard to the origin of this disease among the native population in the areas reported when the condition originated in the Slavic countries of Europe.

HERDENER.

A Case of Rhinoscleroma Cured With Deep X-ray. Um caso de rinoscleroma curado delo raio X profundo.)

Da Silva, Georges, Rev. Brasil. de Oto-Rino-Laring., 9:22 (Jan.-Feb.), 1941.

The patient was a 24-year-old Brazilian woman who had been ill for four years. The first symptoms were cephalalgia and nasal obstruction, followed by an abundant yellow secretion with a foul odor. On examination, both nares were found to be completely obstructed and the mucosa was not retracted by the use of adrenalin. The obstruction was diagnosed as rhinoscleroma by posterior rhinoscopic examination, and was confirmed by an anatomicopathological examination. Roentgentherapy was administered. The first series lasted 50 days and the patient received 2,480 R.U. (160 R.U. each treatment). With 728 R.U. the growth receded and the right nasal passage became functional. At the end of the first series, electrocoagulation was performed to re-establish the normal respiratory function. A second series of roentgentherapy was administered for another 50 days, and the patient received 1,280 R.U.

LICHTWARDT.

Maxillary Sinusitis. Pathological Concepts. (Senitis maxilar. Concepto anatomopatologico.)

Elisbt, F. C. (Buenos Aires), Rev. Argent. de Oto-Rino-Laring. 25: (Jan.-Feb.), 1940.

The author begins with a proposal to change the usage in Spanish of the word sinusitis, to senitis since this latter term has a root more generally in medical usage than the Latin stem of sinus. This change of course refers only to the Spanish nomenclature. He continues by emphasizing that in order to understand the pathology of the maxillary sinusitis and the rational of its therapeutics a clear knowledge of the anatomical pathology of the paranasal sinuses and their neighboring structures is demanded.

The classification of the sinusitides is fundamental in establishing the true diagnosis which is the guide to the treatment.

The author considers that a histopathologic analysis is necessary to be able to differentiate the clinical forms. Further he feels that it is indispensable, for it leads to fewer errors in recognizing the different manner in which the sinusitis reacts to their infection.

The author describes the anatomy and development of the maxillary sinuses. A classification of maxillary sinusitis is given, and histopathologic protocols are presented.

HERDENER.

PHARYNX

Contribution to the Study of Rare Pharyngeal Tumors: Hemangio-Endothelioma of the Hypopharynx. (Contribuição ad estudo dos tumores raros da faringe: hemangio-endotelioma da hipo-faringe.)

Mangabeira, Paulo, and de Toledo, Rui, Rev. Brasil. de Oto-Rino-Laring., 9:267 (July-Aug.), 1941.

The blastomas generally found in the three portions of the pharynx are malignant epitheliomas of various types. The benign tumors are the rarest and are usually papillomas in the oral pharynx or mixed tumors of the salivary gland type in the nasopharynx. The malignant epithelial tumors predominate in the hypopharynx.

A case is presented of what is believed to be the only tumor on record of the vascular endothelium located in the hypopharynx. The tumor measured 6 cm. in diameter and caused difficulty of breathing and swallowing. Upon excision, a histopathological examination revealed it to be a hemangio-endothelioma. The patient died two years later and although no autopsy was performed, death was believed to be due to bronchopulmonary or esophageal metastases.

Lynch observed that in numerous cases of hemangio-endotheliomas of the larynx, they rarely metastasized and complete cures were obtained in most cases. Similar tumors of the tonsils, palatine vein or nasopharynx resulted in metastases to the bronchial lymphatic chain, lungs and liver, with death in two years in all the cases.

These tumors occur in any age group and are treated by surgery, radiotherapy, radiumtherapy, vaccine of Citelli or combined treatment.

LICHTWARDT.

A Rhino-oral-pharyngeoma Cured by Radiotherapy. (Epitelioma rino-oro-faringeo curado por la roetgenterapia.)

Butler, C. B., and Oreggia, J. C. (Montevideo), An. de Oto-Rino-Laring. d. Uruguay 10:1, 1940.

The authors present the history of a case of rhino-oral-pharyngeoma which, following radiotherapy, was complicated by a superimposed diphtheria. However, the infection came on six years following the intensive therapy on the tumor. Less than a year following the serum cure of her diphtheria the patient was admitted for an emergency tracheotomy due to laryngeal dyspnea caused by

adductor immobilization of the vocal cords, and inflammation due to severe arytenoid lesions. A latent chondronecrosis due to the radiotherapy was discovered. The authors therefore point to the importance of watching for a latent radionecrosis in these cases, and the care to be exercised when there is a superimposed local infection.

HERDENER.

LARYNX

Obstructive Laryngeal Dyspnea in Infancy. (Disnea laringea Obstructiva en la infancia.)

Loza, M. G. (Rosario), Rev. Brasil. de Oto-Rino-Laring. 9:201 (May-June), 1941.

Following a short explanation of the physical and bodily forces which cause the characteristic and diagnostic retraction of the suprasternal fossa in obstructive laryngeal dyspnea, the authors emphasize the importance of endoscopy in diagnosis. Endoscopy has been found useful in differentiating a subglottic edema from a suspected primary laryngeal diphtheria.

Various treatments are discussed as they are used in laryngeotracheal obstruction, acute obstructions, and laryngeal diphtheria. In the latter their own method is stressed in which an endoscope is used to facilitate mechanical removal of the false membrane whenever possible so as to improve respiration. In the treatment of subglottic edema we are warned against the dangers of intubation because of its traumatic effect on already weakened tissues. The authors consider tracheotomy too drastic a procedure in cases of subglottic edema, and feel that better results are obtained by the use of oxygen therapy through nasal catheters with local conservative treatment.

HERDENER.

Laryngeal Edema. (Edema laringeo.)

Fernandez, C., Rev. de Otorrinolaring. 1:95 (March), 1942.

The anatomical studies of the larynx and perilaryngeal regions divide this part into three topographical zones—superior, median and lower—which are separated from each other by ligaments and adhesions of the mucous membrane. Experimental studies have shown

that edema develops in the submucous membrane tissue of the larynx, the ligaments and adhesions hindering its passage from zone to zone.

The author reports a case of edema of the inferior layers or subglottic region after a severe case of grippe. A ring infiltration developed below the vocal cords, becoming rapidly worse. After several treatments had failed, X-ray therapy was tried, which brought about an acute crisis of asphyxia. A difficult emergency tracheotomy was performed. It is believed X-ray therapy would be blamed for the asphyxia because of its vasodilator action. Liquids are thus forced into the tissues, which in this case made edema worse and brought on asphyxia. Previous tracheotomy is advisable in case of X-ray therapy of an edematous larynx. Some cases of edema leave permanent sequelae, as in this patient, who, after a year, showed thickening of the mucous membrane which hindered free movement of the vocal cords, causing dysphonia with a peculiar timbre.

AUTHOR.

TRACHEA

Tuberculosis of the Trachea. (Tuberculose da traquea.)

Belfort, Fabio, Rev. Brasil. de Oto-Rino-Laring., 9:261 (July-Aug.), 1941.

Tracheobronchial tuberculosis is a specific infection of the mucosa or submucosa of the trachea and bronchi. The frequency of tracheal tuberculosis is quite high but is little written about because the symptoms are mild when compared with pulmonary and laryngeal tuberculosis. In a study of 1,236 cases of pulmonary tuberculosis, 99 were found to have tracheal tuberculosis and 80 of the latter had laryngeal tuberculosis.

The trachea has never been known to be the primary site of infection. It always accompanies pulmonary or laryngeal tuberculosis. Pulmonary tuberculosis is the primary site, although the possibility of tracheal tuberculosis being such should not be overlooked. The infection infiltrates the mucosa, usually in the membranous portion between the cartilage rings, although the latter may also be involved. When the small tubercles fuse, they are capable of producing ulcerations. Sometimes the cartilage may be perforated, resulting in a tracheoesophageal fistula. Occasionally tuberculomas are formed which may partially or completely obstruct the trachea.

The clinical symptoms of tracheal tuberculosis are few and may not be valid, and the bacteriological examination is of some value; however, the use of the tracheoscope is most satisfactory. Biopsy is an excellent means of differential diagnosis when cancer, syphilis or scleroma is suspected.

Tracheal tuberculosis has been treated with silver nitrate (5, 20 and 25 per cent solutions), lactic acid, electrocoagulation, and roentgentherapy.

LICHTWARDT.

TONSILS

A Case of Cervical Lymphogranulomatosis With an Initial Chancre of the Tonsil. (Un casa de linfogranulomatosis cervical y chancro inicial en la amigdala.)

Mendiola, R. (Guadalajara), An. de Soc. Mex. de Oftalmol. y Oto-Rino-Laring. 16:220 (July-Aug.), 1941.

The author presents this case from an anatomical pathological viewpoint because to date in Mexican publications extragenital localization of this disease has not been reported. All of the histological lesions that characterize the granuloma of Nicolas and Favre were found and the nature of the lesion was proven by the Frei test. The lesion of the left tonsil is reported as having the character of a permanent lymphogranulomatous chancre. In the superior cervical region on the left plaques of micropolyadenitis are described. The case history and physical examination are presented. The skin was normal. The Kahn test was negative and there were no rectal or genital lesions.

HERDENER.

Observations on 765 Tonsillectomies by the Sluder Method. (Consideraciones sobre 765 amigdalectomias de Sluder.)

Espinosa, Rene, Rev. de Otorrinolaring. 1:87 (March), 1942.

Sluder's method is the best for tonsillectomies performed on children, since it is sure, quick and has no other contraindication than too atrophic tonsils which have a fibrous peritonsillar reaction.

Out of 765 patients operated upon the author had only three hemorrhages (0.39 per cent), a smaller amount than after dissection tonsillectomy. In 762 cases he used general chloroform-ether

anesthesia without a single important accident. In the three other cases he used local novocain (1 per cent) infiltration. Notwithstanding accurate technic, he states that every often he could see remaining small pieces of the inferior tonsillar pole attached to His' plica. This was obviated by using a wire sling.

AUTHOR.

TONGUE

Acute Glossitis of the Root of the Tongue—Treatment Plan. (Glositis basica aguda. Su tratamiento.)

Casteran, E., and Montero, J. (Buenos Aires), Rev. Argent. de Oto-Rino-Laring. (Jan.-Feb.), 1940.

Because of their successful management of a recent group of cases, and the somewhat general confusion in the classification of these cases, the authors desire to expand on the subject.

Etiological aspects and their influence on the areas near the lesion are discussed. The symptomatology and the evolution of the acute stages are developed with a warning against ill-advised tracheotomies. The differential points in acute lingual tonsillitis, tonsillar abscess, and glossothyroepiglottic inflammation are brought out.

Mild forms of the above types are treated with astringent gargles, lactic diets, and topical applications. The acute cases are treated by breaking an immunity blockade based on a theory of local immunity, the end of the treatment being directed at impeding the formation of lysins so as to allow the haptenes to destroy the antigens. The local anatomical block to the entrance of the haptenes is caused by the arterial congestion, lymphstasis, and compressed nerve passages due to the inflammation. The authors break the block by bilateral puncture and aspiration of the inferior parapalatine tonsillar space. Anesthesia of the lingual nerve with novocain which is followed by injection into the spaces of an ampoule each of the haptinogenic serum of Mendez. In conclusion, various types of surgical incisions for drainage of the base of the tongue are described, which can be used should the process become too acute for the above method.

HERDENER.

EAR

Early Diagnosis of Arterial Hypertension by Otoscopy. (Hipertension arterial y su diagnostico precoz por la otoscopia.)

Monteiro, A. (Rio de Janeiro), An. de Soc. Mex. de Oftalmol. y Oto-Rino-Laring. 16:65 (March-April), 1941.

Just as ophthalmoscopy has aided in early cases of unknown hypertensives, so does the author feel now that reliability can be placed on his observation first discussed in a previous publication, in which engorgement and accentuated coloration of the vessel along the handle of the malleus was noted in otoscopic examinations in known and unknown hypertensives.

The author refers to his original publication with an account of the original discovery, and to additional presentations at meetings, and vertifications of his findings by other physicians. The use of this examination is suggested when the specialist examines patients who may present other symptoms of hypertension, so that on confirmation by positive findings he may be referred to the clinician as early as possible.

HERDENER.

The Otoneurologic Diagnosis of Encephalic Tumors. Study of 30 Verified Cases. (O diagnostico oto-neurologico nos tumores encefalicos. Estudo de 30 casos verificados.)

Salem, W., Rev. Argent. de Oto-Rino-Laring., 9:53 (March-April), 1941.

The labyrinth is of greatest value in the diagnosis of encephalic lesions; however, this value decreases in cases of marked intracranial hypertension. Isolated tumors of the cerebellum do not appear to produce spontaneous nystagmus, rather the central vestibular tracts must be involved. In cases of intracranial tumors of long standing (two years or more), with progressive clinical manifestations, but without vestibulo-cochlear disturbances, the posterior fossa may usually be ruled out. After rotatory stimulation, the caloric method of producing nystagmus was found to be of most diagnostic value.

LICHTWARDT.

The Physiopathology of the Caloric Test. (Fisiopatologia de la prueba calorica.)

Fernandez, C., Rev. de Otorrinolaring. 1:2 (June), 1941.

The author presents a case of a bullet wound of the right mastoid with severing of the facial nerve and destruction of the labyrinth. The reactions produced in normal subjects by the caloric test are due to the loss of tonus of the examined labyrinth. The liability of this tonus of increasing or diminishing constitutes the labyrinth excitability.

Labyrinthine tonus cannot be measured in normal subjects. In pathologic subjects it is tested through Romberg's past pointing test. Labyrinthine excitability can be tested in normal and pathologic subjects by means of the rotation, caloric, galvanic and pneumatic tests.

AUTHOR.

Some Physical Problems in the Mechanism of Audition. (Algunos problemas fisicos en el mecanismo de la audicion.)

Riccitelli and Franchini (Buenos Aires), Rev. Argent. de Oto-Rino-Laring. 13: (Jan.-Feb.), 1940.

The authors reaffirm in this work the importance of recognizing the vibratory mechanism in interpreting the physiology of hearing. The auditory organ which as an integral part of this mechanism incessantly transforms vibrations of determined longitude and frequency into sensations which in this case result in hearing is no more than a complex machine of transmission and resonance made up for that end by specialized anatomical structures. The authors point to the importance of the physical doctrines of Helmholtz which attribute to the above the simple function of resonance. Likewise they indicate how this is substantiated by pathology and experimental medicine. The authors go into details on the diversity of opinions on the localization of the vibratory process, ending with the concepts of Twold that deny the hypothesis of resonance. After some consideration to the absolute value of the sintonization of the auditory resonators, they refer to the doctrine of the existence of specific energies. Finally Helmholtz' doctrine remains standing. Sound is perceived only when the auditory nerves are excited. Of its transformation nothing is known.

HERDENER.

MISCELLANEOUS

Dietetics in Otorhinolaryngology. (Dietética em oto-rino-laringologia.)

Mangabeira-Albernaz, P., Rev. Brasil. de Oto-Rino-Laring., 9:95 (March-April), 1941.

Dietetics in otorhinolaryngology is in an experimental stage, with exception of the action of most vitamins. Foods required for normal body function are discussed.

Glasscheib considers vasomotor rhinitis an expression of increased blood alkalinity.

Myerson treated a case of nasal polyp successfully by giving 15 drops of dilute nitrohydrochloric acid orally three times daily for two weeks. At the end of that time the polyp was reduced to less than one-quarter of its original size. He believes that carbohydrates are responsible for most nasal disturbances, such as catarrh and various types of nasal discharges.

Myerson and Shurly found a mixture of raw vegetables and orange juice three times daily to be an excellent postoperative diet to stimulate production of granulation tissue.

Calcium intake is of extreme importance. In a study of 4,000 patients, Sherman showed that only two received a sufficient amount of calcium.

Deficiency of vitamin A causes a metaplasia of nasal epithelium, and riboflavin causes lesions and dermatitis of the lips and around the mouth. Symptoms of vitamin C deficiency are gingivitis, hemorrhage, hyperemia of pharyngeal and laryngeal mucosa, and sensation of pressure and pain at the base of the nasal pyramid. Vitamin D deficiency causes ricketic lesions of many of the bony structures of the head.

LICHTWARDT.

Vitamin K in Otorhinolaryngology. (A vitamina K em face da oto-rino-laringologia.)

Suassuna Filho, J., Rev. Brasil. de Oto-Rino-Laring., 9:143 (March-April), 1941.

Vitamin K administered intramuscularly or parenterally constitutes an excellent antihemorrhagic agent in otorhinolaryngology. Noticeable results are seen in cases where bleeding and clotting times are most abnormal. The liver and intestinal function should be

studied in cases with increased bleeding and clotting times not aided with vitamin K. Hemorrhage during operative procedures should be treated immediately with vitamin K to raise the prothrombin of the blood. Nausea, vomiting and toxic effects have not been observed following the use of vitamin K.

LICHTWARDT.

Postoperative Psychoneuroses in Otorhinolaryngology. (Las psiconeurosis postoperativas en otorinnolaringologia.)

Casteran, E., and Montero, J. (Buenos Aires), Rev. Brasil. de Oto-Rino-Laring. 8:488 (Nov.-Dec.), 1940.

The authors feel that since otorhinolaryngology is a specialty essentially surgical in nature due consideration has not previously been given to the possible postoperative psychoneuroses. Physiopathology, somatic alterations with details on angioneurotic edema, and hyperesthesias are discussed with reference to the postoperative course of neurotics following surgical intervention as carried out in the specialty in general. Some interesting points are brought forth with reference to esthetic nasal operations, and the reactions of the patient to the results which never seem to satisfy them completely. Syndromes of anxiety, melancholia, and hypochondriasis in psycheoneurosis following tonsillectomies are reviewed. References are as well made to neurosis with labyrinthine syndromes following ear operations, and to psychoneurosis developing from operative care of laryngeal affections.

HERDENER.

Extradural Abscess of the Posterior Fossa Causing Trigeminal Neuralgia. (Abcesso extradural da fossa posterior causando neuralgia do trigemio.)

Duarte, I., Rev. Brasil. de Oto-Rino-Laring., 8:425 (Nov.-Dec.), 1940.

A 26-year-old patient in the last month of pregnancy suffered from severe trigeminal neuralgia, cephalalgia, otalgia, nausea and vomiting. One month later, myringotomy was performed with no relief. Second and third myringotomies, two weeks apart, were unsuccessful, so the mastoid was opened under local anesthetic. A large extradural abscess was found, almost completely obliterating the lateral sinus. Ten cubic centimeters of yellow, purulent fluid were removed. There followed complete relief from all pain, and cure followed the drainage.

LICHTWARDT.

Osteomyelitis of the Cranial Bones Following the Caldwell-Luc Operation. (Osteomielite dos ossos do crânio.)

Tavares, A., Rev. Brasil. de Oto-Rino-Laring., 9:161 (March-April), 1941.

Cranial osteomyelitis following the Caldwell-Luc operation is extremely rare. Only eleven such cases are recorded in the literature. The infection spreads from the maxillary sinus to the cranial bones by way of the Haversian canals and the venous spaces. The symptoms of cranial osteomyelitis originating in the antrum are similar to those from the frontal sinuses. Prognosis of osteomyelitis following complications in the Caldwell-Luc operation is very poor. All eleven cases recorded proved fatal.

LICHTWARDT.

Some Considerations Concerning the Plummer-Vinson Syndrome. (Algunas consideraciones sobre el sindrome de Plummer-Vinson.)

Segura, Errecat (Buenos Aires), Rev. Brasil. de Oto-Rino-Laring. 8:385 (Nov.-Dec.), 1940.

The authors in an interesting short article review the main points in the literature with regards to this syndrome as brought out by the two cases they observed. Particular emphasis is placed on a short discussion of definitions, symptomatology, comparison of radiologic and esophagoscopic methods of diagnosis, and additional notes on treatment, in the last with special reference to the anemia, dilatation of the esophagus, and vitamin deficiencies.

HERDENER.

Direct Bronchoscopy in Pulmonary Tuberculosis. (Las broncoscopia directa en los tuberculosos pulmonales.)

Acuña, R. T. (Huilpulco), An. de Soc. Mex. de Oftalmol. y Oto-Rino-Laring. 14:237 (Oct.-Dec.), 1939.

The authors review the development in the use of the bronchoscope in pulmonary tuberculosis. A series of cases from the literature are enumerated with a description of the various types of lesions in the tracheobronchial tree. They demonstrate how the method is useful in diagnosis as well as in local therapeutic measures. Some additional comments are made on the important uses of the bronchoscope in pulmonary surgery both pre- and postoperatively.

The authors give the results of 50 patients who were bronchoscoped at the Sanatorium in Huilpulco.

HERDENER.

True Congenital Facial Paralysis. (Paralisis congenita facial verdadera.)

Elisebt, F. C. (Buenos Aires), Rev. Argent. de Oto-Rino-Laring. p. 85 (March-April), 1940.

The author reviews the literature on previously presented cases of this type and emphasizes their rarity.

There is no definitely established etiological pathology for the lesion. In the past the symptoms has been treated, but the results to date offer little hope for cure. Symptomatic treatment as shown by the methods of orthodontia and correction of teeth occlusion has shown some beneficial results. The author presents a case in which these methods were used with marked success. Advice is also given to follow this procedure with facial muscular exercises to hold the ground gained in treatment. Therefore, before submitting a patient to surgery an attempt at cure should be made by consulting a stomatologist. The article is illustrated.

HERDENER.

The Parotid. Removal With Total Conservation of the Facial Nerve. (Parotida—Su extirpacion con conservacion total del nervio facial.)

Finochietto, Marino, Zavaleta (Buenos Aires), Rev. Med. Municipal, Rio de Janeiro 2:790 (Dec.), 1941.

Following a review of the history of the various technics in parotid surgery, the authors state their preference for the Adson method. The various accidental surgical lesions in the removal of the parotid are reviewed. The surgical anatomy of the parotid is discussed with particular reference to further studies they have done on the separation of the parotid into two lobes as was first described by Luschka, and later asserted by Gregoire. The operative technic is minutely described, their modification of the Adson technic being determined by the above anatomical studies. Several cases with variations from the normal are discussed. The surgical steps are illustrated.

HERDENER.

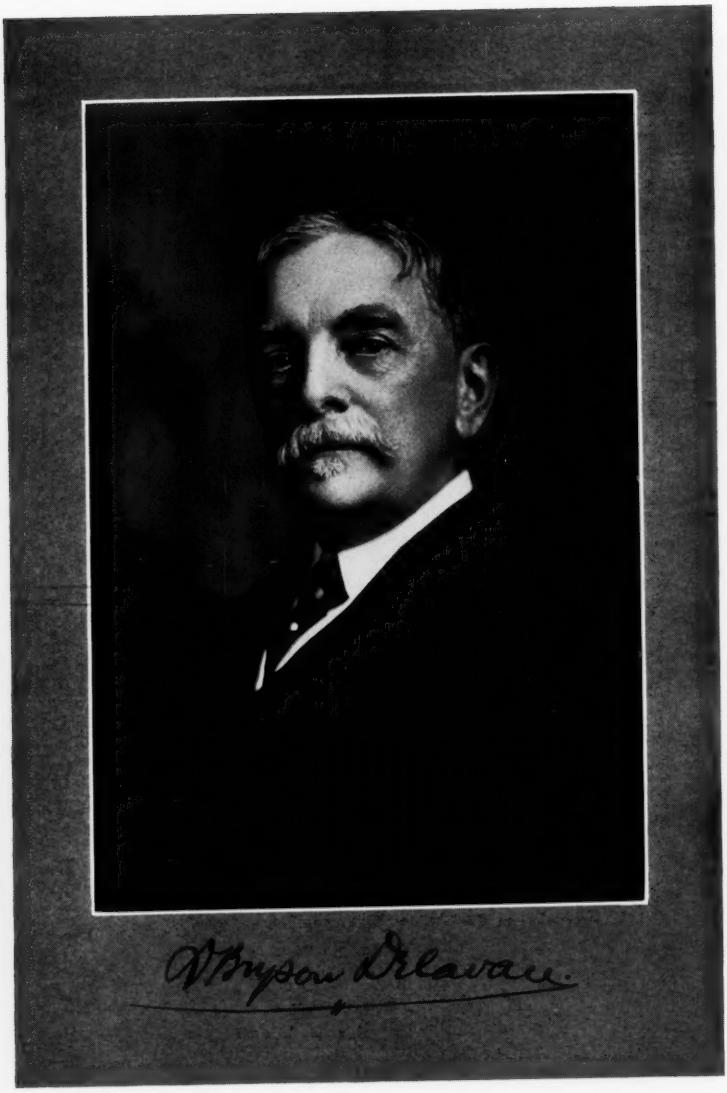
Congenital Cysts of the Neck. (Quistes congenitos del cuello.)

Velasco, R., Rev. de Otorrinolaring. 1:27 (Sept.), 1941.

Congenital cysts of the neck may arise from branchial fissures, the islet of the middle thyroid and thyroglossal duct. There are three kinds of cysts to be found: serous, dermoid and mucoid. The

first are congenital and they are to be found in the sternomastoid region. They are bosselated, badly outlined, renitent and sometimes very large. They cause no pain or functional disturbance. They adhere to the deep layers and especially to the blood vessels. Dermoid and mucoid cysts are smaller and usually well outlined; they appear in early life and localize in the upper front part of the neck. These also adhere to the deep layers, especially the hyoid bone. All congenital cysts of the neck are liable to get infected and suppurate, leaving a fistulous duct difficult to cure. Malignant degeneration can take place as shown in the case presented here. Cure can be obtained by radical removal. Serous cysts can only exceptionally be operated upon before the first year of age. Dermoid and mucoid cysts have to be removed together with a piece of hyoid bone to which they might adhere. Puncture of the cyts is followed by recurrence. Incision is followed by recurrence and fistulization. Radiotherapy does not give good results.

AUTHOR.


W Bryan Delaware

DAVID BRYSON DELAVAN

1850-1942

David Bryson Delavan, long regarded as the Dean of American laryngologists, died at his home in New York City on May 23, 1942, at the age of ninety-two.

This last of the pioneers, though long since retired from practice, participated until the end in the affairs of the Americal Laryngological Association, latterly by correspondence, and a committee report written by him was read to the annual meeting of the Association two days after his death.

Dr. Delavan was born in New York City May 1, 1850. He was the son of Edward Close Delavan and Margaretta Bryson Delavan. His ancestors, of Huguenot, English and Scotch extraction, were among the early settlers of New York City. The name was originally de l'Avant.

He received his early training in private schools and graduated from Yale University in 1872 and from the College of Physicians and Surgeons in New York City in 1875, on that occasion receiving Honorable Mention for an essay on "Tuberculous Laryngitis". While an interne at Charity Hospital he obtained permission of the authorities to gather all the cases of laryngeal diseases into one ward which became known as the Laryngological Ward, and was probably the first of its kind in this country.

After entering the practice of medicine Dr. Delavan became first assistant to Dr. George M. Lefferts, the leading American teacher of laryngology of his day, then Professor at the College of Physicians and Surgeons, a post which he retained for twenty-one years.

In the course of the next few years he made many connections and held several positions, among them the Curatorship of the Museum at the New York Hospital, assistant to the pathologist Dr. George Livingston Peabody, and attending physician to the Work House Hospital.

In 1887 he was made Professor of Laryngology and Rhinology at the Polyclinic Medical School and later a trustee and member of the Board of that institution.

Soon after its inception in 1875 Dr. Delavan became a member of the New York Laryngological Society founded by Dr. Clinton

Wagner, and the first laryngological association in the world. This was the forerunner of the American Laryngological Association which came into being five years later. He was president of this association in 1893 and again on the occasion of its fiftieth anniversary in 1928. In 1931 the Association bestowed upon him the De Roaldes Medal, its highest honor, "in recognition of many distinguished contributions to the advancement of medical science."

He was also a Fellow of the Royal Society of England, Corresponding Fellow of the British Laryngological Association, the American Medical Association, the American College of Surgeons, and numerous local societies.

He was also associated in various capacities with the Russell Sage Institute, the Grenfell Association of America, the Scenic and Historic Preservation Society, and the American Museums of Natural History and of Art. He was a member of the University and the Yale Clubs of New York.

Even before the beginning of the Century Dr. Delavan became absorbed in the subject of laryngeal cancer, and wrote numerous papers on various phases of malignancy in the upper respiratory tract. In later years, as Chairman of the Committee on Control of Cancer, he made comprehensive annual reports to the American Laryngological Association.

Slight of figure but distinguished in his bearing he was a familiar figure at the meetings of the various societies, beloved for his courtesy and his loyalty to the traditions which he had done so much to build. These meetings he attended until a very few years preceding his death.

He was married June 14, 1899, to Miss Marion Rumsey of Saint Louis who died Dec. 21, 1939. He is survived by a daughter, Mrs. William Randolph Moore of Greenwich, Conn., and two grandchildren, William Randolph Moore, Jr., and Marion Rumsey Moore.

A. W. P.

VINCENZ MUELLER

1865-1942

An old friend has gone to his reward.

With the passing of V. Mueller the medical profession has lost a staunch colleague, a benign adviser in the technical problems which beset the inventor (alas, often only the *re*-inventor) of surgical instruments, and withal a jovial companion.

This man was a genius. He enjoyed taking infinite pains. He gloried in perfection. No instrument too tiny, no creative idea so vague that it did not compel his attention. A year after some conversation there would appear one morning, in the mail a contraption devised by V. Mueller embodying the desired principles—and practical, more often than not. He was the silent partner in the development of many an instrument now bearing some famous name.

Mr. Mueller was born in Liptingen, Germany, Jan. 21, 1865. As a boy he was apprenticed to a manufacturer of instruments in Tuttlingen. Having a desire to live in one of the English speaking countries, he went to London where he worked for about five years as an instrument maker, then came to Chicago in 1893 as a foreman for the firm of Frank and Kratzmueller. About a year later he was married to Miss Florence Heydenreich, whose people were instrument makers in London.

In 1896 Mr. and Mrs. Mueller established a small business in the Medical Center of Chicago and until the time of Mrs. Mueller's death, some twelve years ago, she continued to be active in the business. Mrs. Mueller was known to many of the older specialists as she always attended the various meetings of the eye, ear, nose and throat specialists when exhibits were held.

After Mrs. Mueller's death, her husband traveled extensively. He made friends easily and it was his custom to visit the hospitals and surgeons wherever he went.

Some five years ago Mr. Mueller decided that he would retire and became Chairman of the Board of his Company. With his energy, however, actual retirement was impossible. His associates aver that he worked harder than ever. He had been troubled with severe neuritis for years and often said that the pain was less when he could keep on working, which he continued to do almost until the time of his death, the result of a coronary attack.

One child, Mrs. G. W. Wallerich, survives him.

His memory will long be green.

A. W. P.

Books Received

Directory of Medical Specialists Certified by American Boards, 1942.

Pp. xvi + 2,495. Morningside Heights, N. Y., 1942, Columbia University Press. (Price $7.00.)

A great many new names have been added to the previous list and the volume is larger than its predecessor by some 900 pages.

Most important: The various sections have been indexed by states at the top of the page, greatly facilitating the location of names.

Information, complete; typography and binding, the best.

Roentgen Treatment of Infections.

By James F. Kelly, Professor and Director of the Department of Radiology, Creighton University School of Medicine; Attending Radiologist, Creighton Memorial St. Joseph's Hospital, St. Catherine's Hospital and Douglas County Hospital, Omaha, and Mercy Hospital, Council Bluffs, Ia.; with the collaboration of D. Arnold Dowell, M.D., Assistant Professor of Radiology, Creighton University School of Medicine; Assistant Attending Radiologist, Creighton Memorial St. Joseph's Hospital, St. Catherine's Hospital and Douglas County Hospital, Omaha, and Mercy Hospital, Council Bluffs, Ia. Pp. 1-432, with 122 illustrations and 25 tables. Chicago, 1942, The Year Book Publishers, Inc. (Price $6.00.)

This is an absorbing monograph on a subject which touches the otolaryngologist only remotely at the present writing. Mastoiditis is discussed at some length but in this disease the authors accord radiation such faint praise as to leave the impression that it has little worth. One does not find any mention of otitis externa except as this may come under "Boils and Carbuncles" or "Infections of the Upper Half of the Face". They feel that "X-rays will localize [infections of the upper lip, cheek and in and on the outside of the nose] in a large percentage of cases and the prognosis, although guarded, is definitely better with X-ray than with surgical treatment" on which score they will encounter no argument. The discussion of sinusitis covers less than a page and gives little encouragement for the use of X-ray in this field also.

From a general standpoint and in fields in which radiation has its chief sphere, the book is well written, informative, and well put together.